

**CLINICAL PÆDIATRICS**  
(THE BABY)

# CLINICAL PÆDIATRICS (THE BABY)

EDITED BY

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## FOREWORD

In the past the care of the new born infant has been in the hands of the doctor or nurse attending the mother in her confinement. For this reason some aspects of neo natal care have received insufficient attention and many serious conditions have been overlooked. The remedy has been found in a special study of these problems and has produced that branch of medicine called *Pædiatrics*.

This subject can be divided into three periods, that of the infant, the child and the adolescent. Each of these is distinct, and presents its own problems and diseases. Most present day text books endeavour to cover the whole of the vast field, invading the domain of the obstetrician on the one hand and that of the physician on the other. The result is either a bulky work, too unwieldy for the student, or an unreadable precis useless as a book for reference.

I therefore welcome this text book on the baby from the pen of Dr. Collis and other members of the Dublin Medical Schools. The book has been wisely confined to a study of the baby, and all its normal and abnormal conditions have been fully considered. The use of modern methods and scientific advances applicable to the treatment of infantile conditions are excellently described.

The value of co-operation between the obstetrician and the pædiatrician is fully borne out by the lowering of the infantile mortality at the Rotunda Hospital since the formation of a special infants' department some years ago. These good results are due to the practical application of the methods recommended in this text book.

In this book, recent work and established rules of procedure for dealing with the baby during the neo natal period are discussed and the correct practical methods stated. The work, however, is by no means confined to this period, but deals with the baby during the first years of life in health and disease. Certain chapters, such as that on tuberculosis, contain much original work and thought and will, I hope attract attention. The sections of ophthalmology, otology, orthopædics infectious

diseases etc., have each been written by a specialist on the subject. Some of these subjects have never before been approached from quite the same point of view, *e.g.*, few people realise the importance of ophthalmology in the baby (15 per cent. of adult blindness is caused by trouble at this period). Hence these special sections will serve as invaluable references for doctors confronted with any such special problems which they may meet when dealing with small children. The doctor will be able to ascertain what to do, who should do it, and when it should be done, be the problem congenital cataract, cleft palate, club foot, ectopia vesicæ etc. For these reasons I believe the book will have a wide appeal for students requiring simple practical methods, general practitioners needing an up to date reference work, and to all who are engaged in obstetrical practice.

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DUBLIN

# CLINICAL PÆDIATRICS

## (THE BABY)

### SECTION I

#### CHAPTER I

C J MCSWEENEY

#### INFECTION, IMMUNITY AND PREVENTION OF WARD INFECTIONS

(Infection—The Source and Transmission of Infection—Immunity—  
Prevention of Ward Infections—Aseptic Technique in Nursing)

**Infection** The invasion of living tissues by pathogenic agents constitutes infection. Pathogenic agents may be bacteria (*e g*, diphtheria bacilli, streptococci), protozoa (*e g*, malaria parasites) metazoa (*e g* intestinal worms) or viruses (*e g*, of chicken pox, small pox, mumps, measles). Infection implies pathogenicity, but the converse is not true. Pathogenic organisms may be present in the body without infection taking place. The carrier state affecting those immune to diphtheria, or long recovered from enteric fever, is not an infection. A carrier is important epidemiologically but not clinically. His effects are communal rather than individual—infection on the other hand primarily concerns the welfare of the individual whose tissues are invaded.

The virulence of an organism is a measure of its capacity for invading living tissues. This does not mean that only virulent organisms are able to cause severe constitutional disturbance and death. Organisms of relatively low virulence may produce severe symptoms by means of the toxins they excrete into the tissues. The diphtheria bacillus is an example of an organism with but slight invasive powers which acts in this way. The constitutional disturbance in diphtheria is entirely due to the toxins absorbed from the membrane wherein the organisms are localised.

Toxins separable by filtration of cultures from their related

bacteria are called *exotoxins*. When inoculated into the animal body an *exotoxin* always causes characteristic signs and symptoms. *Exotoxins* are excellent antigens for the production of the corresponding *antitoxins*. Examples of pathogenic agents producing *exotoxins* are the causative organisms of diphtheria, tetanus and botulism. All three have low invasive powers but are capable of elaborating toxin from the local tissue effects they cause.

The organisms producing toxins which are not separable from their related bacteria by filtration of cultures constitute a much larger class. These *endotoxins* differ from *exotoxins* in almost every particular. They are not specific in their effects on animal tissues, the lesions produced vary and they are only poorly *antitoxinogenic*. The limited therapeutic value of anti-bacterial as compared with *antitoxic sera* is attributable to these factors. It is probable that those organisms which produce only *endotoxins* depend for their pathogenic action more on their invasive than on their *toxigenic capacities*. Included in this group—to mention three organisms producing widely divergent anatomical lesions—are the *pneumococcus*, the *meningococcus* and *B. typhosus*.

A few organisms produce both *exo* and *endo toxins*. The scarlatinal group of *hemolytic streptococci* belongs to this class. The soluble *exotoxin* of the scarlatinal *streptococcal group* is the antigen with which horses are immunised in the commercial production of *scarlatinal antitoxin*. This *exotoxin* is also used in the intradermal test for susceptibility to scarlet fever and in active immunisation against that disease.

The pathogenic effects produced by the same organism may vary considerably at different ages. Thus an upper respiratory infection in an infant may spread rapidly to invade the lungs and the resultant *streptococcal broncho pneumonia* may be quickly fatal. Such an event is exceedingly rare in healthy children or adults although in old age the same sequence may be observed as in infancy. Again the *pneumococcus* is highly virulent for healthy young adults but produces only slight constitutional disturbance when it causes *lobar pneumonia* in children. Likewise the degree of toxicity exhibited by organisms varies with age. Thus the toxin of the diphtheria bacillus is especially prone to attack the cardiovascular system and peripheral nerves of children often with fatal consequences but these effects are much more rarely encoun-

tered in adolescents and adults even when allowance is made for the lessened attack rate at later ages

The exact explanation of these phenomena is not known

### The Source and Transmission of Infection

The three routes by which infection is transmitted—*inhalation ingestion and inoculation*—have one common source viz, a human being or animal harbouring the causative organism of the disease. The source of the great majority of human infections is either a case or a carrier—exceptions being those diseases of animals which are transmissible to man *e.g.* anthrax tetanus rat plague bovine tuberculosis undulant fever and Weil's disease

The source of infection may not be immediately obvious but in the common infectious diseases of urban life (diphtheria scarlet fever measles whooping cough chicken pox mumps cerebrospinal fever) is almost invariably another human being—either a case (diagnosed or missed) or a carrier

Inanimate objects (books clothes bedding etc.) are often blamed for sporadic cases of infectious disease but are seldom responsible. The causative organisms of the common communicable diseases rarely survive for long the stony ground of inanimate material for none of them is spore bearing. Although most infections are spread directly from person to person occasionally the route is indirect *e.g.* through the medium of milk or water. In this way the range of attack may be lengthened and the field broadened considerably. A diphtheritic dairymaid in the country can transmit diphtheria to hundreds of city dwellers the few Klebs Loeffler bacilli which she has added to the milk pail having multiplied to millions in the churn during transit. Similarly the pollution of a mountain stream with enteric organisms from the faeces or urine of a case or carrier may cause a typhoid outbreak by contaminating the water supply of a town fifty miles away. Nevertheless the source of infection is ultimately human and the milk or water but the vehicle by which the infection is transmitted from person to person. Again the louse in typhus and the flea in human plague bridge the gap between infected persons and susceptible victims but the original source of infection is a human being suffering from typhus or plague. An indirect method of transmission which is important in

hospitals is the transference of germs from case to case on the hands of attendants. When there has been a departure from aseptic nursing technique children with enteritis impetigo contagiosa otitis media etc. can infect other children in the ward by this method of manual transference.

Inhalation is the commonest route by which the endemic infections of these islands are transmitted—examples are the common cold influenza pneumonia scarlet fever diphtheria whooping cough cerebrospinal fever pulmonary tuberculosis and small pox. Transmission of infection by this route is not the simple process of inspiring an atmosphere in which pathogenic agents are floating. Infection by inhalation would probably never be effected except for the fact that minute infection laden droplets are being constantly propelled into the air around an infectious person. Coughing and sneezing greatly facilitate the propulsion of infected droplets and no doubt the occurrence of these catarrhal symptoms in measles whooping cough and the common cold explains the high attack rate observed in these infections.

Ingestion is a route which is of special importance in early childhood. Infective enteritis abdominal tuberculosis as well as dysentery enteric bacterial food poisoning undulant fever and in tropical countries cholera are examples of infections transmitted by swallowing.

Inoculation plays a small part in the causation of infections in this part of the world where malaria yellow fever plague and now also typhus are unknown but erysipelas and pemphigus neonatorum are still met with and both of these infections are inoculated through the abraded cuticle. So also in all probability is Weil's disease.

When infection breaks out in a children's ward the blame is usually attributed to visitors who are in consequence exiled from the infected wards for weeks or months. Ward infections are much more commonly endogenous than exogenous. The visiting of sick children in hospital by over anxious and sometimes fussy relatives may be undesirable for many reasons but the danger of infection being introduced by this channel has probably been over emphasised. The ideal arrangement under which adults may be allowed to visit children in hospital is to permit them to look through a glazed peep hole about 3 inches in diameter which is let into the ward door. There is then no danger of their transmitting upper respiratory infections and

as the child cannot see the observer, there is none of the distress and emotional disturbance which the arrival and departure of parents is apt to provoke

Before concluding this brief resumé of infection and its routes of transmission it may be permissible to mention two common misconceptions which appear still to survive. The first of these is the relation of *defective drains* to infectious disease. A house with choked drains is no more likely to be visited by infectious disease than one not so affected. a choked drain causes a bad smell and is a nuisance which ought to be remedied but it does not of itself cause diphtheria or any other infectious disease. To expose the drainage system when a case of diphtheria occurs in a house benefits no one except the plumber. it is far more rational to endeavour to trace the human nose or throat from which the causative organism emanated—unless this be done a carrier in the household or among the victim's playmates will continue to cause diphtheria even though the drains have been seen to. Another popular fallacy about infection concerns *peeling* in scarlet fever. Peeling of itself does not imply continued infectivity. Haemolytic streptococci do not choose the dry inhospitable medium of a piece of cuticle for survival when there are available warm moist mucous membranes in the nose and throat. Scientific experiment as well as the accumulated experiences of many fever hospitals have shown that desquamated scales play no part in the spread of scarlet fever. Myths of this kind die hard and it is the duty of the practitioner when opportunity offers to help to eliminate them from the minds of his patients and their relatives.

**Immunity** Immunity or the faculty of resisting disease depends upon a variety of circumstances or conditions which can only be touched upon briefly here. Broadly speaking immunity to a particular disease implies previous experience of its causative organism or some biological product thereof. Certain races and some individuals tend to be insusceptible to particular infections and this phenomenon has in the past been called natural immunity but it is very doubtful whether the continued use of this term is justifiable. Natural immunity so called whether racial or personal is relative and can be overcome by starvation exposure anxiety states neglect of personal hygiene or other adverse circumstances inimical to the individual. A transient passive immunity to most infectious diseases except pertussis is undoubtedly possessed by infants

during the first six months of life, whether this be due to protective substances transmitted to the infant by the maternal blood (and possibly breast milk) or to the sheltered life which young infants enjoy as compared with other children is not known with certainty—probably both factors are at work.

Acquired immunity is the series of defence mechanisms by which the human body withstands invasions by pathogenic agents. It varies in degree. It may be achieved passively by inoculation of protective substances present in the blood of another animal but this immune state is of brief duration and is probably not absolute for longer than two or three weeks. Thus 1 000 units of diphtheria antitoxin or 3 000 units of scarlatinal antitoxin will protect an individual from the clinical consequences of exposure to diphtheria or scarlet fever, but a month later he will be just as likely as an uninoculated person to contract these infections.

Immunity may be acquired actively by an attack of the disease, this immunity is often complete for a period but it is probably never absolute permanently. The immunity conferred by an attack of measles is possibly the most complete and most lasting of any produced by an infectious disease. Many people enjoy an immunity to a particular disease as a consequence of repeated doses of infection—too small to cause a clinically recognisable form of the disease but sufficiently potent to stimulate the defence mechanisms of the body to resist actual invasion of the tissues by the organism. This process of latent immunisation is probably very active in urban centres and is no doubt responsible for the higher degree of herd immunity possessed by town dwellers as compared with rural inhabitants.

Active immunity is also acquired by the inoculation of certain substances which are known to stimulate the tissues to produce antibodies capable of resisting invasion. An example of this is the degree of immunity to diphtheria produced by the inoculation of diphtheria prophylactic. The individual here produces circulatory antitoxin sufficient to neutralise diphtheria toxin when injected into the skin, and in the great majority of cases sufficient to withstand an attack of clinical diphtheria. Starting with a small dose of scarlatinal toxin and gradually increasing it at intervals, a high degree of immunity may also be acquired to scarlet fever. Vaccines (e.g. T A B) act in the same way by stimulating the tissues to produce protective

substances which prevent or attenuate the clinical manifestations of infectious disease

In recent years another method of acquiring immunity has been practised. By the use of the immune serum of another individual, a child exposed to measles may be completely protected from the effects of that exposure. A potent immune serum is rich in antibodies and if given within four days of exposure passively immunises the child, who escapes all symptoms and signs of measles. This protection is fleeting (probably not longer than two weeks in duration). If the serum be withheld until the fifth, sixth or seventh day after exposure, it will not protect the child from attack but will cause the course of the illness to be much milder. This process of sero attenuation of measles is also achieved by using the citrated whole blood of one or other parent. Attenuation of the attack does not interfere with its immunising effect and the child recovered from a modified measles is probably as immune from measles as a child who has just managed to survive a severe attack. It will be obvious that in the case of immune serum, administered during the first few days after exposure, a passive, transient, though complete immunity is conferred by the serum, whereas administration after the fourth day results in a partial immunity which allows a mild attack of measles to produce an active and permanent immunity to the disease. The latter procedure, viz., sero-attenuation is, of course, the method of selection when dealing with delicate children for whom an attack of unmodified measles might have disastrous results. Passive immunisation with measles serum is probably never indicated except as a means of counteracting the accidental introduction of measles into hospital wards.

## THE PREVENTION OF WARD INFECTIONS

Modern pædiatrics is practised under many difficulties. Not the least of these is the legacy of large wards in which sick children must be nursed, another is the serious overcrowding of these wards, which the steadily increasing demand for skilled treatment in hospital has brought about. A third is the failure of lay administrators to realise that a children's hospital requires to be generously staffed with nurses if ward infections are to be reduced to a minimum.

Infants and young children are exceedingly susceptible to

respiratory and intestinal infections, having contracted these conditions they are very apt to die of them. The same organisms which cause a common cold in a healthy child may evoke a fatal catarrhal pneumonia in a weakly infant, while the admission of a case of infective enteritis to a sick children's ward may directly or indirectly contribute to the deaths of half a dozen other children.

New hospitals or extensions to existing buildings ought to be constructed on the small ward plan—preferably in single cubicles separated by glass partitions with a balcony in front on to which cots can be wheeled in fine weather. Existing large wards should, as circumstances permit be divided up into smaller units by the erection of glazed partitions preferably taken up to the ceiling, but certainly not less than 7 feet high.

Where cubicles are not available, beds should be distant at least 12 feet from one another and preferably arranged parallel rather than at right angles to the wall.

All sick children ought to be nursed on ' barrier or bed isolation ' principles the nurse wearing a separate overall and thoroughly washing her hands after dealing with each case. As upper respiratory infections and skin diseases spread rapidly in a children's ward, often with dire consequences nurses who have colds sore throats discharging noses or ears or septic fingers should be rigidly excluded from handling sick children. The nurse who prepares distributes and administers the ward meals should have no responsibilities in regard to napkins bed pans and urinals. A generous provision of washable overalls, wash hand basins bed pan sterilisers and boiling sinks for cooking and feeding utensils, is essential if aseptic nursing is to be carried out efficiently.

## THE ASEPTIC TECHNIQUE IN NURSING

It has to be admitted that a rigid observance of ' bed isolation ' or ' barrier nursing ' for all patients is not at present practicable in many children's hospitals. The following modified ritual should, however, be possible without the need for a material increase in nursing staff —

(1) All patients with any obvious infection (e.g., any form of catarrh of the respiratory system, running nose discharging ears pharyngitis, tonsillitis, enteritis, impetigo or other septic skin condition) should be " bed isolated ". Such a patient is

best treated in a cubicle or on a roofed verandah. If neither of these facilities be available his bed ought to be screened—glass partitions 7 feet high being placed around three sides of it—and the distance between this bed and the clean beds in the ward must never be less than 12 feet.

(2) Each bed isolated patient must have a complete set of requisites for his own use—feeding crockery, bed pan, urinal, toilet articles, thermometer, etc.

(3) A nurse attending on an isolated patient must wear an overall covering her uniform and don rubber gloves when doing dressings on such patients. The gown and gloves are kept at the bedside and are reserved exclusively for the isolated case.

(4) After attending to a bed isolated case the nurse having shed the gown must thoroughly wash her hands in ordinary soap and water and use a nail brush.

(5) If the isolated case is suffering from an upper respiratory infection, a nasal discharge, otorrhœa, pharyngitis or tonsillitis, the nurse should wear a cellophane mask when attending to the patient. Gauze masks unless frequently sterilised are a danger rather than a protection.

## CHAPTER II

KERRA REDDIN

### INFANT MORTALITY AND CHILD WELFARE

(Comparative Tables on Infant Mortality—Summary Education Breast Feeding, Free Milk, Development of the Ante- and Post-natal Clinics Home Visiting, Neo-natal Mortality Rates Organisation of Child Welfare Centre, General Routine in Child Welfare)

A STUDY of the English Registrar General's mortality figures (1871-1906) reveals the interesting fact that though there was a marked decline in the general mortality rate which includes adults, the infantile mortality rate did not show a similar decline

Table I

Year	General Death Rate per 1 000 Population	Infantile Mortality per 1 000 Births *
1871-75	21.9	103
1881-85	18.7	139
1891-95	18.5	151
1901-05	16.0	138

\* Deaths under one year comprise infant mortality rate which is calculated as the proportion of deaths of infants under twelve months of age to every 1 000 births for the same year

Now if the infant mortality rates from 1906 onwards are studied in Table II it can be seen that a steady fall has taken place from that year onwards. This fall has coincided with the institution of Child Welfare Schemes and the Notification of Births Act.

Under this Act all births are made notifiable to the Medical Officer of Health of the city or county where the birth takes place. Stamped post cards on which date and place of birth are to be filled in are supplied free to all practising midwives in the area. There are special large forms for hospitals and institutions. Notification is compulsory. The object of the Act is to facilitate home visitation by health visitors, by furnishing the Medical Officer of Health with the time and place of birth.

Infant Mortality Rate (England and Wales)

Deaths per 1,000 Births Registered

Table II

Year		Year	
1906	132	1921	83
1907	118	1922	77
1908	120	1923	69
1909	109	1924	75
1910	105	1925	75
1911	130	1926	70
1912	95	1927	70
1913	108	1928	65
1914	105	1929	74
1915	110	1930	60
1916	91	1931	66
1917	96	1932	65
1918	97	1933	64
1919	89	1934	59
1920	80	1935	57

There can be no doubt that the education of the mother by health propaganda, the teaching of mothercraft, and home visiting by nurses under Child Welfare Schemes have played a big part in this fall in the infant mortality, though it cannot be denied that improved housing, dust proof road surfaces and standardisation of milk production have been contributing factors

Turning now to Irish statistics the following table is instructive —

Table III

Year	Urban	Rural	Dublin City
1923	99	50	125
1924	104	55	125
1925	97	53	119
1926	110	56	127
1927	99	56	123
1928	91	56	102
1929	93	58	106
1930	90	56	97
1931	88	58	94
1932	95	59	100
1933	85	54	83
1934	78	55	80
1935	87	57	94

If the above table is studied it is seen that the urban death rates are higher than the rural which suggests that density of population, conditions of living, nutrition, lack of housing accommodation etc., in the larger towns are the determining factors in the higher urban rate. However, in spite of the bad living conditions of the poor in Dublin there has been a marked decline in the urban infantile death rate here, again coinciding with the development of the maternity and child welfare work.

Some of the most important aspects of a child welfare scheme may be summarised as follows —

(1) **Education** A large proportion of infantile deaths are attributable to maternal ignorance and the young mother's aptitude to follow the advice of elder female relatives versed in the ancient lore of ignorance and superstition which still surrounds baby management. Hence advice for young mothers has been the first function of our Child Welfare Clinics. Here the right attitude towards the whole process of child bearing has been taught. Pregnancy should be regarded as a physiological state not a pathological condition.

(2) **Breast Feeding** No factor has had more influence on the fall in the infant mortality rate than the campaign to encourage breast feeding. Every mother has been encouraged to feed her baby and the danger of weaning during the hot weather, when infantile diarrhoea is epidemic, impressed upon her. In Chapter IX the many questions concerned in breast feeding are fully dealt with, hence it is only necessary to say here that one of the most important functions of the Welfare Centres should be to encourage breast feeding by advice and propaganda.

(3) **Free Milk** The system of supplying good free milk to babies of unemployed families has proved a great boon to the poor and has undoubtedly helped in the reduction of the infantile mortality rates.

(4) **The Development of Ante- and Post-natal Clinics in the Maternity Hospitals** Though much still remains to be done the development of these clinics has been a potent factor in preventing the disasters of pregnancy and assisting the mothers' health during the post partum period, and therefore secondarily in reducing the infantile mortality rate.

(5) **Home Visiting** The visiting of all maternity cases in the city during the second week after confinement by a health visitor has proved a most valuable measure. One of the

most important functions of the health visitor is to establish contact with "the first baby mother," advise her, and bring her into the clinic

(6) Co-operation The central Child Welfare Centre has proved a focus where much of the health work for infancy and childhood in the city has been co-ordinated, e.g., the children's hospitals, ante and post natal clinics in the maternity hospitals, the orthopaedic hospitals, the orphanages, homes, etc., together with the Municipal Child Welfare Clinics

Much remains to be done, but these facts and figures show without doubt that the child welfare movement has proved its worth to the community

Now let us examine the neo-natal mortality rates —

Comparative Table showing Death Rates in Infants under One Month Old per 1,000 Births

Table IV

Year	Dublin City	England and Wales
1923	35.90	31.90
1924	39.62	33.00
1925	35.65	32.30
1926	40.90	31.90
1927	40.80	32.30
1928	35.67	31.10
1929	34.56	32.80
1930	34.42	30.90
1931	32.76	31.60
1932	31.02	31.60
1933	30.72	32.20
1934	28.04	31.30

Table IV shows the neo-natal death rates in Dublin and England and Wales from 1923 to 1934. It can be seen when these figures are compared with the general infant mortality rates (given above) over the same period that whereas the general infant mortality trend has been steadily downwards during these years, the infant mortality rates of the neo-natal period have remained almost constant. The Dublin figures show more fluctuation than those for England and Wales, but this is due probably to the numbers being smaller in the former. We may now ask what is the cause of this fact and why have not the factors which have reduced the general infantile mortality had a similar effect on the neo-natal

mortality.<sup>2</sup> The answer to these questions is undoubtedly that babies during the neo natal age are not generally under the care of the Child Welfare Clinics and children's physicians. They are supervised nominally by the obstetrician during the first week or ten days after birth, after this period they are rarely seen by a doctor till they are brought to the welfare centres, which is seldom before the third or fourth week. A clear policy is necessary here, and careful co-operation is essential between the maternity hospital service the child welfare department and the children's hospitals. Clearly provision for all non infectious neo natal conditions *e.g.*, grave jaundice hemorrhagic disease of the new born prematurity etc. should be made in the maternity hospitals where special accommodation should be provided for such babies born both in the hospital and on its district. All infectious cases (*e.g.*, respiratory alimentary skin or other infection) should be treated in the children's hospitals and a system of the closest co-operation developed between the maternity and child welfare service and the hospitals. Such co-operation can only be brought about by the development of social service (almoner departments) in the hospitals, whose duty should be to keep in touch with the maternity and child welfare health visitors and the welfare centres.

### Organisation of Child Welfare Centre

**Premises Required** If at all possible these should be on the street level and should consist of the following minimum accommodation —

- (1) Pram shed
- (2) Waiting room
- (3) Weighing room
- (4) Doctor's room
- (5) Lavatory
- (6) Isolation room (small)

The size of the rooms will depend on the possible attendance at the clinic. We find on an average in Dublin that the waiting room requires to accommodate up to 140 but in smaller rural areas 25 to 40 is an average attendance. It is better to err by allowing for a larger attendance, for as the work of the scheme progresses there will always be an increase in numbers. The floors should be of some impervious material, if possible, and

easily cleaned, old floors should be covered with linoleum. The rooms should be bright, airy and well heated. Open fires, with appropriate guards, will suffice when central heating is not available.

A complete card index system of the mothers attending ante and post natal clinics, and of the infants and children up to five years, should be kept in each clinic.

**Staff Required** Two trained nurses, with some help from local voluntary workers can manage an average clinic, the senior nurse giving the health talk and supervising the running of the clinic. The other nurse, or nurses, are engaged in weighing, dressing and undressing the babies. The distribution of such simple nutrients as cod liver oil can be carried out by the voluntary helpers.

**Furniture Required** Waiting room a sufficiency of forms, or chairs, should be provided for the waiting room, a black-board for lectures, a table for demonstrations with a doll baby clothes and bath, and a large play pen for unruly toddlers.

**Weighing Room Requirements** Table, wall thermometer, washing facilities and weighing machines, preferably of the bar and balance type.

A separate paper napkin should be provided for the weighing of each baby, and the room must be kept at a temperature of 60° to 65° Fahrenheit. Plaster for strapping umbilical hernia, some dry dusting powder, low nursery chairs for the mothers undressing their babies, and separate baskets for the babies' clothes should be provided.

**Doctor's Room** should contain table chair, examination couch (adult size) and a small urine testing outfit.

**Clinic Activities** may be summarised as follows

(1) Weighing the babies

(2) Advice from nurses

(3) Advice from the doctor in attendance

(4) Treatment of a very few minor ailments

(5) The sale of nutrients, such as cod liver oil, Virol, emulsion, at a low price, to mothers who can pay, and free distribution to those who cannot pay.

(6) Group teaching, health talks, etc

(7) Special classes cookery, sewing, etc

(8) Home visiting

Expectant and nursing mothers are seen and advised together with children up to five years of age. The expectant mothers

are referred to the nearest maternity ante natal department. We feel sure that in this lies the best way of organising ante and post natal work and child welfare. Through the homely atmosphere of the welfare clinics it is possible to teach mothers expectant and nursing with greater effectiveness than can be done at special hospital ante natal clinics though the actual examinations must be done in the latter.

In the Child Welfare Clinic mothers are advised on infant feeding, minor ailments are treated and more serious cases referred to those hospitals with accommodation for infants (e.g. from one to five years of age patients seen with orthopaedic and other deformities such as squint, enlarged tonsils, ear discharges, etc. are referred to the special departments of the nearest hospital).

**General Routine in Welfare Clinic.** Having left her pram in the pram shed and having removed from it all shawls, covers, etc. the mother proceeds to the waiting room. If she has other children with her she brings them to the waiting room where the toddlers may best be left in the play pen. If the weighing room is full the mother waits her turn in the waiting room and listens to the health talk given by the senior nurse. The mother then goes to the weighing room, is given a basket and undresses her baby. If she has no shawl she is given a blanket to wrap the baby in. The child is weighed naked. If it is losing weight or if the mother wishes to consult the doctor about the baby she then waits her turn to do so. Otherwise the baby is dressed and the mother is free to go home.

**Weighing.** In a Child Welfare Clinic the weighing of babies is a very important part of the work. Where possible the babies should be weighed naked. Weighing provides —

- (1) An index of the child's progress
- (2) A warning of commencing failure of breast feeding
- (3) A standard for infant feeding

There is no other means so simple and accurate of gauging the normal progress of the infant. The average healthy baby doubles its weight in the first six months and gains on an average 4 to 6 oz. a week.

**The Health Talk.** Group teaching is being increasingly employed in child welfare work. Practical demonstrations of bathing and dressing should be given. These can easily be managed by the purchase of a cheap doll and bath with a set of baby clothes. No health talk is of any value that lasts over

ten minutes, it should be short and simple, and questions should be encouraged on the part of the audience. The lecture must *not* be read from notes, and must be given in the form of a talk rather than a lecture. Too much emphasis cannot be laid on the desirability of making the mother feel that she can easily get help and advice on the handling of her baby and that the atmosphere of the clinic and its staff is sympathetic.

**Home Visiting.** The "Health Visitor," as the nurse working under the Notification of Births Act is called, visits the mother's home after the tenth day, offers her advice, stresses the importance of continuing breast feeding as long as possible, and refers her for specific medical advice and assistance to the nearest Child Welfare Clinic. Her further duties are to enquire into the home conditions of the family and to advise accordingly.

**Qualifications of Health Visitors.** (1) The nurse who is to act as health visitor must possess her certificates of general training and her C.M.B.

(2) She should be old enough to advise a mother, yet not too old.

(3) She should be kind, tactful and good-natured, having an attitude of mind indicating that her function is to help those mothers and children under her care, not merely to fill in forms and pile up statistics.

To keep nurses keen on their work and up to date in their ideas, nothing is more helpful than a weekly or fortnightly lecture on such subjects as :—

- (1) Difficulties met with in the working of the scheme.
- (2) New and improved methods of dieting, etc
- (3) Relation to other municipal schemes, such as housing, etc.
- (4) An occasional physiological résumé or clinic on cases of disease met with in the course of the work.

## CHAPTER III

W R F COLLIS

### GROWTH AND DEVELOPMENT DURING FIRST YEAR OF LIFE

(Weight Table Weight Height and Circumference of Head—General Appearance—The Head—The Teeth—The Senses—Speech)

EVERY infant's growth and development is different and the widest range is found among normal healthy infants. Hence the facts and tables given below must be regarded merely as an aid when judging any particular case and it must be remembered that the figures are composed of averages.

**Weight** The average weight of the new born male infant is  $7\frac{1}{2}$  lb. If the infant is below  $5\frac{1}{2}$  lb it should be regarded as a premature baby even when the mother's dates do not suggest this. During the first week there is usually an initial loss of weight due to the passage of meconium and the usual delay in the appearance of breast milk. But this should be only temporary and should be followed by a quick rise after the third to fifth day so that the birth weight is again reached or passed by the end of the first week. Large babies usually lose more weight than small ones during the first week.

Normal infants should double their birth weight by the sixth month and treble it by the end of the first year.

Below is given a table showing the average weight height and circumference of the head of boys and girls during the first year of life.

Age	Weight pounds		Height inches		Circumference of Head in inches	
	Boys	Girls	Boys	Girls	Boys	Girls
Birth	7.50	7.10	20.6	20.5	13.9	13.5
6 months	16.0	15.7	27.4	25.0	17.0	16.6
12	20.0	19.8	29.0	28.7	18.0	17.6

**General Appearance** At birth babies have a red skin, some become jaundiced (see p 51) but all should assume the normal pink and white appearance by the end of the first month. The skin should be elastic if picked up between the finger and thumb. In cases of dehydration the loss of elasticity of the skin is one of the first signs (i.e. when picked up it fails to spring back into place and tends to lie in folds on the subcutaneous tissues).

The normal infant has a thick layer of subcutaneous fat which fills in the hollows of the body lines. In cases of malnutrition this fat is lost rapidly and the infant becomes hollow cheeked and emaciated in appearance, often with great rapidity. By the sixth month the muscles should feel firm and the baby should be able to sit up for a moment or two. By nine months he should be able to sit up without difficulty. By fifteen months he should be walking. Great differences in the rapidity of muscular development are found in normal healthy infants and too much stress must not be laid on the absence of any one of the above stages of growth.

In cases of vitamin D deficiency (i.e. rickets) though the baby may appear fat and well covered he will not be able to sit up, on examination his muscles will be found to be soft and his ligaments lax, while the bones will be unusually soft and pliable.

**The Head** The condition of the head is also an aid when ascertaining the development of an infant. The closure of the fontanelles is somewhat variable and depends to some extent on the moulding of the head during labour. Usually the posterior fontanelle should be closed at three months. The anterior fontanelle is not closed however, before eighteen months. In rickets the bones of the head remain soft and the fontanelles open. Also in cases of hydrocephalus the fontanelles and sutures will remain open the cranial contents will protrude and the circumference of the head will be larger than normal, in progressive cases the circumference will rapidly increase out of all normal proportions.

**Teeth** The development of the teeth begins in early uterine life and hence, as we shall see later, the mother's diet is of paramount importance if correct dentition is to take place. The teeth of children born of mothers who have been on a deficient diet will tend to be deformed at dentition and to decay rapidly afterwards.

There are twenty milk teeth their date of eruption is very variable even in healthy children on a well balanced diet

The following table gives the approximate dates when the different teeth may be expected to appear —

Lower central incisors	6- 9 months
Upper incisors	" 12
Lower lateral incisors	12 21
First molars	15-21
Canines	16-24
Second molars	20-30

By two and a half years the baby should have twenty teeth

**Special Senses** At birth the eyes are unable to fix an object though by the end of the first week they will often follow a bright light The muscles of the eyes only gradually learn to act together Individual objects begin to be recognised by the end of the first six months

At birth infants are said to be completely deaf Hearing commences in about forty-eight hours and by the end of the first week it should be normally acute Individual sounds may be recognised by the third month

Taste in the infant is highly developed though the interpretation of this stimulus by the infant's brain may be quite different from that of the normal adult *e.g.* an infant will take a sour acid milk with as much relish as a sweet milk provided he is used to it If the mixture is changed he will often object This suggests that the stimuli of taste are not as fundamental as those of pain and that they are based on habit

Pain and Tactile Sensibility are present at birth but the reflex action which follows is not appreciated by the higher centres till about the third month Hence it is possible to perform such minor operations as circumcision without an anæsthetic shortly after birth without causing mental trauma

**Speech** The commencement of speech in the normal baby is very variable and backwardness in this respect up to the second year should not cause anxiety as to the child's mental development If no attempt is made to speak by that time the child should be brought to a pædiatrician for complete examination Most children begin to say single words by the end of the first year and are able to say short sentences by the end of the second year

## SECTION II

### CHAPTER IV

W R F COLLIS

#### MANAGEMENT OF THE NORMAL INFANT DURING THE FIRST WEEK

(General Measures—Temperature—Weight—Colour—Urine—Meconium—The Cord—Prepuce—Mouth, Eyes and Nose—Sleep and Crying—Feeding—The Test Feed—Method for Increasing Flow of Breast Milk—Typical Weight and Temperature Charts )

As soon as the infant is born its mouth should be cleared of mucus with a sterile swab and the eyes cleansed. As soon as respiration has commenced the infant should be wrapped up and placed in a specially prepared and warmed cot till it can be attended to further. It is of the utmost importance that it should not be allowed to cool and the above steps are an absolutely necessary routine.

As soon as possible the baby should be bathed in warm water and thoroughly cleansed. It is most important that the bath water should not be allowed near the baby's eyes and that as soon as the bath is over a drop of silver nitrate 1 per cent. be put in each eye. (*Note* Care must be taken that the drop actually falls on the conjunctive and not merely on the lid (see p 403) ) After the bath the child should again be carefully wrapped up and placed in the specially warmed cot. If born in hospital, it should be removed as soon as possible from the labour ward and placed in charge of the sister of the puerperal ward. *In all cases it is necessary to take special care that the child does not cool down between birth and arrival in the ward.*

Whether in hospital or the district in a nursing home or private house, a definite routine should now be followed if the many disasters which may occur during the neo natal period are to be averted.

(1) **Temperature** A morning and evening temperature chart should be kept during the first week. Such a record may in some cases be necessary for an even longer time. The informa-

tion thus obtained is of the greatest importance. Pyrexia may denote cerebral irritation due to transmitted maternal toxæmia, cerebral œdema following prolonged labour, intracranial hæmorrhage or dehydration fever. An alarming fall in temperature is noted if the infant is allowed to cool unduly after its bath. It is not unusual to find a subnormal temperature in premature and weakly children; this may be the first sign of abnormality in an apparently healthy infant. It is remarkable that even now in many maternity hospitals and nursing homes no temperature and weight chart is kept for the baby and that many midwives are surprised if asked to keep them.

(2) **Weight** Every baby should be weighed at least three times during the first week, preferably every day. Unless complementary feeds are given before the breast milk appears, the majority of babies will lose a few ounces during the first three days. Birth weight should be regained almost without exception by the end of the first week. Many healthy infants whose mothers have an ample supply of breast milk will gain several ounces during this time. A steady loss of weight up to the seventh day signifies either an insufficient supply of breast milk or some pathological condition in the child.

(3) **Colour** The normal baby has a pink or red appearance for the first forty-eight hours. This gradually changes to the paler hue of the normal older child. In about 30 per cent of normal infants some degree of physiological jaundice develops. This type of jaundice is never severe and soon disappears. There are, however, several grave forms of jaundice which occur during this period. Their early recognition is of the utmost importance if the child's life is to be saved (see p. 51).

In some forms of congenital heart disease and sometimes in atelectasis, constant or intermittent cyanosis may appear during the neonatal period.

(4) **Urine** Urine should be passed normally during the first twelve hours and from that time on at regular intervals. It is not always easy to tell whether a small quantity of urine has been passed if the nurse has let the nappy dry. There is no need for anxiety unless the bladder is distended to well above the symphysis pubis. Usually placing the infant in a warm bath will cause reflex emptying of the bladder. If no urine has been passed after forty-eight hours it must be withdrawn by catheter. This is, however, rarely necessary.

(5) **Meconium** During the first twenty-four hours meconium

should be passed five or six times. It is dark green in colour. The motions do not assume their normal yellow appearance till the third or fourth day.

(6) *The Cord* The cord should be dressed daily and dusted with powder. The usual method is to bring the cord out through a hole in a piece of dry sterile gamgee tissue keeping this in place by an abdominal binder. If kept dry sterile separation occurs without trouble leaving no raw surface. Should any weeping surface remain it will clear up if touched once or twice with a silver nitrate stick. Unless daily aseptic dry dressings are applied the umbilicus may become infected and this may lead to death from septicæmia (see p. 59).

(7) *Prepuce* The prepuce of every male child should be examined. If it cannot be drawn back over the glans perform circumcision (*vide* p. 357) before the third or fourth week. If the prepuce is sufficiently loose it should be pulled back daily, the glans and urethral orifice being cleansed with a swab and sterile water.

(8) *Mouth, Eyes and Nose* The mouth should be cleansed out two or three times a day with a swab of sterile cotton wool dipped in warm boiled water. Similarly the eyes should be sponged night and morning. The nose may be kept clean with swabs moistened in warm water or some mild disinfectant such as glycothymoline or boric lotion.

(9) *Sleep and Crying* The baby should not be nursed in the same bed as the mother lest he come to expect continuous attention and the body warmth of the mother. Overlying is a definite danger. The baby should be in a cot at some distance from the mother's bed and fed at regular hours. The napkins should be changed regularly and the child wrapped warmly without interference with the movements of the hands, legs and respiratory muscles. Relatives and visitors should be instructed to avoid breathing and speaking over the child or lifting it out of the cot for droplet infection is a very real danger.

The temperature of the room should be kept between 60° and 70° F.

If this suggested routine is followed the infant will sleep most of the day and night during the first week. If he cries and does not sleep something is wrong, he is either hungry, thirsty or uncomfortable. Hence when a baby cries seek the cause. Should crying be persistent some pathological condition must

be suspected and the child should be undressed and carefully examined

(10) **Feeding** Most important of all is the problem of feeding. During the first week it is necessary to have a very definite schedule. Eight hours after birth the baby should be put to the breast and encouraged to suck for five minutes at each breast. From now onwards he should be put regularly to one breast every three hours i.e. at 6 a.m., 9 a.m., 12 noon, 3 p.m., 6 p.m. and 10 p.m. During the first three days the baby will receive only *colostrum* and will require boiled water (which is best sweetened with sugar) at regular intervals. Colostrum (the plasma like secretion which the breasts produce before the establishment of lactation) is of the greatest importance to the new born infant. It contains certain antibodies necessary for the infant's defence against disease in the first few months of life. Hence in spite of the fact that the infant may not receive more than  $\frac{1}{4}$  oz. per feed during the first day regular breast feeding is very important. Sugar (5 per cent) and boiled water must be given in ample quantities till the breast milk appears so as to supply the infant with the necessary amount of fluid. As we have seen the normal baby tends to lose weight during the first few days. If this loss continues and the baby cries and sucks his hands it is probable that he is not getting sufficient breast milk, and a test feed should be performed forthwith.

**Test Feed Method** The baby is weighed in his clothes then given the breast and then weighed again. The difference in weight will be the amount of breast milk received. One weighing may give inaccurate information however as the amount of milk available varies during the day. Hence it is always necessary when performing a test feed to weigh at least three consecutive feeds and take the average.

A normal baby should receive  $2\frac{1}{2}$  oz. breast milk per 1 lb. body weight per day. If the test feed shows that the supply of breast milk is insufficient certain measures may be adopted in the hope of increasing its secretion.

The following instructions may be issued to mothers in such circumstances —

(1) Give both breasts at each feed. allow the child to suck for five to ten minutes at each breast. It is important that the order in which the breasts are given to the baby should be changed each

time so that the breast which is sucked first at one feed will be sucked second at the following feed

(2) Express any milk left behind and give from spoon or bottle This should be done after the baby has finished at the breasts Quite a surprising amount of milk may be left behind by the baby

(3) Local Applications to the Breasts (a) *Massage*

(b) *Hot and cold sponging*

(4) General Health Measures for the Mother

(a) *Diet* Order plenty of good food especially eggs meat fish and vegetables *Avoid overfeeding* Plenty of fluid and one pint of milk daily are beneficial

(b) *Sleep* Suggest going to bed as early as possible and rest by lying down for one or two hours during the early afternoon

(c) *Exercise* Some mild outdoor exercise is good but should never lead to tiredness

(d) As far as possible exclude all worries

It is important for the mother to realise *that the vigorous suction of the infant at the nipple is the most important factor*

If in spite of these measures the amount of breast milk does not increase at once complementary feeding (see p 83) must be commenced

If the baby cannot be breast fed at all by reason of lack of development of the breast tissues failure of secretion maternal tuberculosis etc artificial feeding should be begun without delay Most children tolerate cow's milk For this reason it should be recommended for all normal infants who cannot be breast fed It is preferable to the many proprietary foods at present so widely advertised The latter are more expensive than ordinary cow's milk often less satisfactory and generally unnecessary The majority of infants will tolerate a 2:1 milk water and sugar mixture from birth (see p 91) If the infant is weakly it is best to begin with a mixture of equal parts of sweetened cow's milk and water during the first few weeks The problem is dealt with fully in the chapter on artificial feeding

Below are given two charts which show how valuable a temperature and weight record may be during the neo natal period

Fig 1 gives the temperature and weight curves of a perfectly normal baby—the temperature varies between 97.6° F to 98.4° F There is a loss of weight during the first three days but the birth weight is regained on the sixth day, and thereafter continues to rise

Fig 2 shows similar curves of a baby with a persistent low

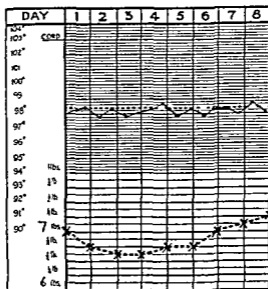


FIG 1

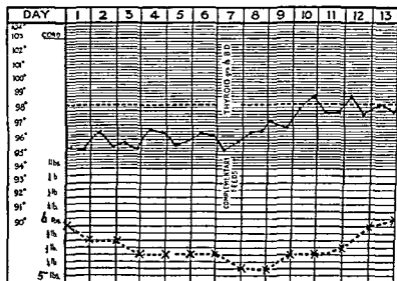


FIG 2

temperature (95° F to 96° F) and a steady loss of weight up to the seventh day. A test feed was then performed. As the baby was found to be getting only an average of 1 oz per

breast feed, complementary feeds were given. At the same time thyroid gr.  $\frac{1}{10}$  B.D. was given to bring the temperature up to normal (see p 33). These measures proved successful, the temperature and weight curves at once improving—the temperature reaching normal by the tenth day and the birth weight being regained on the twelfth day.

Since the introduction of these charts the supervision of the babies in the puerperal wards of the Rotunda Hospital has been greatly simplified. It is possible now to walk round the wards and pick out immediately any case which is not behaving normally by glancing at the charts.

## CHAPTER V

W R F COLLIS

### THE PREMATURE INFANT

(Causes—Heat and Thyroid Administration—Feeding—Nursing—Infection—Anæmia Summary of Routine—Ultimate Prognosis)

There is no satisfactory definition of a premature infant hence the arbitrary rule is taken here that all babies with a birth weight of 5½ lb and under should be regarded as premature whether their mothers dates correspond or not. This difficulty of classification makes it almost impossible to compare the mortality figures for premature infants from different centres. Hence their mortality figures vary between 30-60 per cent. One fact however is clear the death rate among premature infants is exceedingly high and prematurity forms the highest single cause of death in the neo natal period. Can this state of affairs be remedied? The author feels that it can and that no field of medical therapeutics has been more neglected than that of the treatment of prematurity. Indeed it is not too much to say that over 20 per cent of these deaths could be prevented if doctors and nurses acquainted themselves with the requirements of its modern therapy. A carefully worked out routine for all cases is essential which must then be followed meticulously. Below such a method is given in detail. Before however speaking of the treatment of the premature infant it is necessary to discuss for a moment the problem of prevention.

*The causes* of prematurity are multiple and fall within the province of the obstetrician rather than the pædiatrician. Prevention consists in ante natal care specially directed towards keeping the mother healthy. No doubt measurements and every preparation for the actual confinement are an essential part of the work of any ante natal clinic but equally important is the general care of the mother during her pregnancy. If her general health is maintained at a high level by a properly balanced diet and careful hygiene she will avoid half the pitfalls of this period and the infant will be provided with

sufficient calcium and phosphorus for bone formation iron for its blood and general dietetic constituents for the construction of its other tissues while ample vitamins will ensure that growth proceeds along correct lines and all the disasters of pregnancy, premature birth among them will be less liable to occur

The principles upon which our method is founded may be summarised as follows —

**A Heat** The maintenance of a normal body temperature is the most essential single factor in the treatment of prematurity. These infants sometimes appear to have an only partially developed heat regulating mechanism also their basal

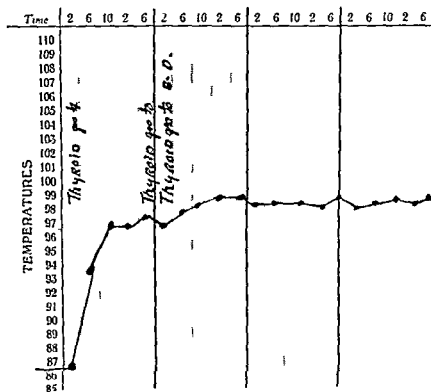


FIG 3

This baby was born of a mother who had undergone a long and difficult labour. He breathed almost at once after birth and in consequence no special treatment was accorded him. He was washed and placed in a warm cot surrounded by hot water bottles. Some hours later he was noticed to look grey and was immediately sent down to the nursery and there his temperature taken. It is interesting to note that notwithstanding this very low temperature the special measures adopted in the nursery for his resuscitation were successful and that the child recovered. Birth weight 4½ lb

metabolism often appears to be below normal. It is pointed out in all the text books that they lose heat very quickly. This is quite true but what is not generally appreciated is that they often fail to produce enough heat from their body metabolism to maintain a normal temperature.

On p. 29 is given a chart of a baby of  $4\frac{1}{2}$  lb. admitted to the nursery of the Rotunda Hospital.

In the author's opinion heat loss due to external causes is of less importance in these cases than subnormal temperature due to failure of metabolism.

Another essential point is brought out by the above case. Action must be prompt. The infant cannot be put in a cot and left till someone has time to look after him. Immediately after he is born measures must be adopted to maintain his temperature. The cord should be ligatured at once. It is a fallacy to suppose that the baby gains by the extra blood he may obtain from the placenta if the cord is not divided at once. He has more blood than he needs and waiting exposes him to loss of heat. The author feels that failure on the part of many obstetricians and midwives to appreciate this principle is a potent cause of neo natal mortality both amongst premature infants and other weakly babies.

No premature infant should be bathed in water. Instead, he should be cleansed in warm olive oil and immediately wrapped up in a warm blanket. In the maternity hospital the infant should be removed at birth to a special infants' nursery where a trained staff should take over his management.

Various measures to prevent *heat loss* are used in different hospitals. Our routine may be stated as follows: the baby should be rubbed with warm olive oil, he should then be placed in a gamgee or loosely knitted garment care being taken to cover the back and sides of the head (see further details below in summary of routine treatment). He should then be placed either in a room with conditioned air (e.g., constant wet bulb—65 per cent. humidity—and a temperature of 80° F.) or in some form of specially heated cot. The simplest of these is a cot with protected sides (so as to cut off draughts) and either hot water bottles beneath and around the child or some form of electrical heating (e.g., electrically heated blankets, an electric bulb slung above from a surgical cradle which has been covered with blankets) or the child may be nursed in some patent type of incubator. But these measures alone are

often insufficient to keep up the baby's temperature and certain measures must also be adopted to stimulate his metabolism

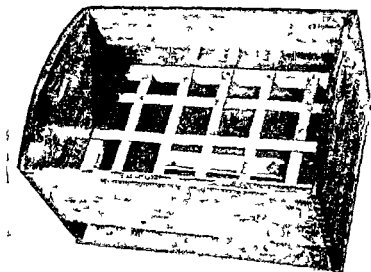


FIG. 4

Simple Incubator Cot used in Rotunda Hospital modified from Dr. Allen's used in Belfast and made here by the Laxlip Woodworking Co. Inside measurements  $22\frac{1}{2}$  inches long by  $15\frac{1}{2}$  inches wide sides  $11\frac{1}{2}$  inches high ends  $13\frac{1}{2}$  inches high with handles cut in them for lifting the cots. A space of 4 inches is left along one side of the cot as seen in the photo to give access to the lamp. A 60 watt tubular electric lamp (striplite) about 7 inches long is fitted in a reflector about 13 inches long and the latter is screwed to the bottom of the cot. About 2 yards of flex is connected to the striplite fitting for plugging in at points on a wall. About  $4\frac{1}{2}$  inches above the bottom of the cot is fitted a removable mattress formed of zinc strips criss crossed on an oak frame. When fitted up with mattress blankets etc. the cot is kept at a constant temperature of  $80^{\circ}$ – $85^{\circ}$  F. These incubator cots have been found satisfactory for nursing the majority of premature and weakly infants and some twenty to thirty are now in use in the Hospital. They also have the advantage of being easily portable and can be used in private houses or nursing homes if required.

Various methods were tried out in the Rotunda nursery (e.g., the administration of memformon (œstrin) pituitrin and various stimulants) without noticeable success till Pritchard's method of giving large doses of thyroid was adopted. He recommends giving thyroid to all premature and weakly infants to stimulate their growth and metabolism. We have found that thyroid gr  $\frac{1}{4}$  (Burroughs Wellcome)\* can be given without toxic effects to almost any premature baby.

\* Thyroid gr  $\frac{1}{16}$  (B.W.) = thyroid gr  $\frac{1}{16}$  (B.P.) approximately

This will often raise the body temperature as much as  $10^{\circ}\text{F}$  in four hours. Our routine practice is now to give thyroid gr  $\frac{1}{4}$  (B W) to all premature infants admitted to the nursery with a subnormal temperature and to follow this by thyroid gr  $\frac{1}{16}$  (B W) twice a day for the following three to four weeks.

When it is realised that thyroid gr  $\frac{1}{4}$  (B W) given to a premature infant of 4 lb is equivalent to approximately thyroid gr 25 (B W) to a fully grown man, it becomes clear that the normal thyroxin production of these infants must be very deficient. Certain experimental evidence supports this view and Cooper\* claims that the thyroid gland does not secrete thyroxin during the first three weeks of extra uterine life. Our work supports this view. The normal baby is born apparently with enough thyroxin to last over this period of inactivity. Premature babies have not this store to draw upon and hence tend to show signs of thyroid deficiency immediately after birth.

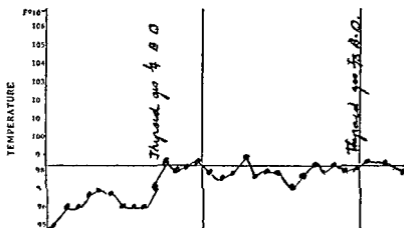


FIG. 5

The above chart demonstrates the value of thyroid administration. If studied it will be seen that the baby's temperature varied between  $95^{\circ}\text{F}$  and  $97^{\circ}\text{F}$  for four days, then thyroid extract (gr 0.25) was given for a week and the temperature at once rose to normal and was maintained there. At the end of a week the dose of thyroid was reduced to gr 0.1 twice a day. Birth weight 4 lb 12 oz.

Not only, however, does the administration of thyroid help to maintain the body temperature of these infants but as the following graph shows it also stimulates growth and development in no small degree.

\* Cooper Human Endocrine Glands etc Oxford Med. Publ., 1925

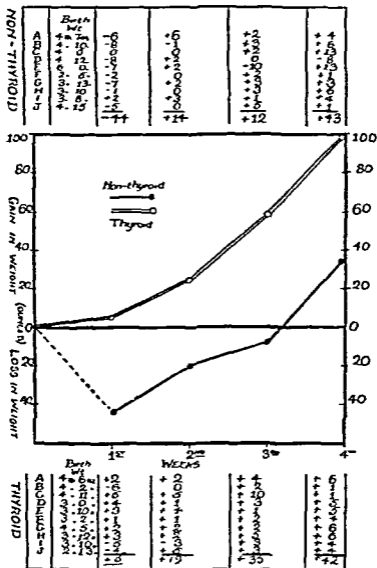


Fig. 6

Graph Demonstrating Weight Curves of 10 Premature Babies on Thru'd and 10 Controls.

The double line represents ten babies on thyroid treatment and the single line ten controls. At the end of the first week the thyroid group are seen to have gained 5 oz., whereas the

controls have lost 44 oz. It is not till the fourth week that both curves become parallel and the weight gain in the two groups equal.

(*Note* This experiment was performed before we adopted the 5½ lb rule for premature babies but this in no way invalidates the principle demonstrated.)

**B Feeding** The next most important factor in the management of premature infants is the problem of feeding. Whenever possible breast milk should be obtained. If this is impossible from the mother as is often the case a wet nurse should be employed. In all maternity hospitals a breast milk service should be organised. In certain cases it will be necessary to increase the caloric value of the breast milk, and this can best be done by the addition of sugar in the form of lactose or dextrin maltose. If breast milk is unobtainable a milk formula with a high caloric value is best. Whole milk with an additional 5 per cent carbohydrate or dextrinised cereal is often highly successful. If the infant appears unable to tolerate this a condensed milk (*e.g.* Nestlé's) or an acid milk (*e.g.* Lacidac or ordinary prepared lactic acid milk) may be tried.

The problem of how to feed the baby is one about which it is impossible to be dogmatic except in so far as the total amount that should be taken in each twenty four hours is concerned. Here it is necessary to follow some definite routine which the nurse can have to hand as a printed table. For this purpose we use the following table.

### Feedings (Breast Milk) for Premature Infants during the First Week

#### Daily Quantity of Feeding, Ounces (Holt and McIntosh)

Weight of Baby	1st Day	2nd Day	3rd Day	4th Day	5th Day	6th Day
2 lb (1 000 gm.)	Milk 2 Water 2½	Milk 2½ Water 2	Milk 3 Water 1½	Milk 3½ Water 1	Milk 4 Water ½	Milk 4½ Water 0
3 lb (1 500 gm.)	Milk 3½ Water 3½	Milk 4 Water 3	Milk 4½ Water 2½	Milk 5 Water 1½	Milk 6 Water 1	Milk 7 Water 0
4 lb (2 000 gm.)	Milk 4½ Water 4	Milk 5 Water 4	Milk 6 Water 3	Milk 7 Water 2	Milk 8 Water 1	Milk 9 Water 0
5 lb (2 500 gm.)	Milk 6 Water 5	Milk 7 Water 4	Milk 8 Water 3	Milk 9 Water 2	Milk 10 Water 1	Milk 11 Water 0

If the amount to be given in the twenty four hours is kept constant, the times of feeding and the amounts and methods of administration can well be left to the nurse. Some premature babies will be able to take the breast, others can suck a specially small teat, others must be fed by dropper or spoon, and to some the fluid can only be administered by gavage. Again the amounts taken per feed will vary in every case. Hence no definite rule can be made either regarding the size of each feed or the frequency of feeds.

**C. Nursing** This brings us to possibly the most vital factor of all in the management of these tiny infants—the nurse. Ultimately the success or failure of all our therapeutic measures will depend upon the nursing. If the nurse has a “flair” for babies, and is efficient and conscientious, the babies will thrive. If she is slack or “bored,” they will die. Hence it is the author’s rule when he finds himself working with a good nurse to give her full charge of the case, only directing the main lines to be followed while leaving the details to her discretion.

**D. Infection** Premature infants are particularly prone to pick up infection and if they do having little immunity to disease, they are apt to die very quickly. The commonest route for infection to take is that of the respiratory passages, hence these babies must be guarded against droplet infection in every way. Visitors must not be allowed in the nursery in the hospital, and the babies must be isolated in the nursing home from the endearments of their female relatives.

The vexed question of masks is a problem to which there seems no solution. Undoubtedly if all the nurses and visiting doctors wear masks the chance of infection is lessened, but the habitual wearing of a mask greatly reduces the nurse’s efficiency and the pleasure she takes in her work. Probably a compromise is the best solution. If any of the staff has any upper respiratory infection, she should be put on her honour to report it at once. If the infection is slight a mask may be worn till it has disappeared, but if severe the nurse must be sent off duty.

Fingers are also a very potent source of infection. No nurse with a septic finger should be allowed in the nursery. The strictest precautions must be taken when nursing any child with an infected skin.

**E Anæmia** Premature babies are specially prone to jaundice during the first two weeks. This is as a rule merely a slightly exaggerated form of the usual physiological jaundice (see p. 51). The hæmolytic often passes the normal limit, however, and leaves the baby anæmic. This may be due to the fact that the red blood corpuscles tend to show an increased fragility in premature infants.

Later also a secondary anæmia often develops in these cases due to lack of storage iron. Hence prophylactic measures to combat anæmia should be adopted in all cases of prematurity. Raw egg yolk has been found to be very useful in this respect, and is now given to all our premature babies as a routine. When an anæmia develops in spite of this iron is administered in some assimilable form (see p. 190).

The following suggested routine is based upon the above principles and is submitted here in the hope that it may be of service to any doctor or nurse who is suddenly confronted with the problem of a premature baby in surroundings not specially organised for the management of such cases.

### Suggested Routine for Premature Babies

(1) As soon after birth as possible take the baby's temperature per rectum.

(2) Weigh. do not leave naked while weighing but keep covered with a specially warmed blanket of known weight.

(3) Sponge with warm olive oil.

(4) Dress as quickly as possible. In the maternity hospital where numerous premature babies are being treated and where there is an abundance of hospital supplies a gamgee jacket which can be changed daily is probably the best garment to put next the skin. This may be prepared by cutting a piece of gamgee long enough to reach from the shoulder to the hips and wide enough to meet at both sides. A circular hole is cut for the neck with a short slit down the front so as to enable the head to be pushed through. This is secured with a loose binder and a good wool coat with long sleeves used outside it. The napkin must be small and of light material. A hood of gamgee should be made for the head as well. Finally the infant is wrapped in a light blanket wound sufficiently freely so as not to impede respiration.

In private practice if a loose knitted garment with a hood

can be obtained, it may be preferred to gamgee, as it is less likely to become sodden with olive oil

(5) Place in a specially heated cot. If no form of incubator is obtainable use the following method. Line the cot with some warm material to shut off draughts. Wrap the baby in blankets not sheets. Place two rubber hot water bottles covered with flannel and a blanket lengthwise, under the baby, turning the necks of the bottles towards the foot of the cot, as in this position they can be easily removed and, should leakage occur, the water will flow away from the baby.

(6) Now give by mouth water  $\frac{1}{2}$  oz glucose  $\frac{1}{4}$  drachm and thyroid gr  $\frac{1}{4}$ . If unable to suck give by spoon if unable to swallow, by gavage.

(7) Watch colour carefully, and at the end of two hours again take the temperature. If the baby's general condition and temperature are not now satisfactory give a rectal coffee saline  $\frac{1}{2}$ -1 oz (1 pint water sodium chloride  $\overline{5i}$  coffee  $\overline{5iv}$ ) and brandy  $\overline{Mv}$ - $\overline{x}$  hyperdermically into the buttock.

(8) Commence feeding two hourly. The following times for feeding are usually satisfactory 2 a.m. 6 8 10, 12 2 4, 6, 8, 10 p.m. (*e.g.* ten feeds in twenty four hours). If not anxious for feeds give 2 drachms of normal saline ten minutes before each feed.

(9) Give thyroid gr  $\overline{3v}$  B.D. for three to four weeks.

(10) On the seventh day give Allenbury's dextrin maltose, 1 drachm to 2 oz, and cease giving glucose.

(11) In most cases give  $\frac{1}{2}$ -1 oz rectal coffee salines four hourly for first two weeks, then give normal rectal salines till the infant no longer retains them.

(12) On the seventh day commence giving raw egg yolk,  $\overline{Mu}$  daily, increasing thereafter slowly till  $\overline{5i}$  is given daily during fourth week.

(13) Once a day remove baby from cot sponge with warm olive oil and change clothes. (*Note* This must be done in a very warm air temperature.)

**Prognosis** "Will my baby grow up strong and normal?" is a question asked by the parents of all premature infants. If the baby is not diseased or malformed in any way, the answer is "Yes providing special care is taken during the first year."

Premature babies are more prone than ordinary infants to catch infection, to suffer from deficiency diseases and get anaemia.

But if they are carefully guarded from infection and their diet arranged so as to assure them a feed not only of higher caloric value than that of the normal infant but also one containing an ample supply of vitamins and sufficient iron in some assimilable form they should develop into as satisfactory a child as any normal infant

## CHAPTER VI

W R F COLLIS, H L PARKER, F GILL

### ASPHYXIA NEONATORUM, ATELECTASIS AND BIRTH TRAUMA

(Asphyxia Neonatorum • Diagnosis and Treatment—Mouth to Mouth Method Nasal Obstruction in the New-born. Atelectasis Neonatorum : Diagnosis—Treatment. Pneumonia Treatment. Injury at Birth • The Brain—Injury to Peripheral Nerves—Injury to Spinal Cord Depressed Fractures of the Skull Bones Other Birth Fractures Clavicle—Humerus—Femur. Ruptured Blood Viscus—Treatment. Ruptured Bowel—Treatment)

#### ASPHYXIA NEONATORUM

ASPHYXIA neonatorum falls into the province of the obstetrician, but no work on infant pædiatrics would be complete without a discussion on this subject, which recently has been receiving special attention. In the older obstetrical text books asphyxia neonatorum is divided into "blue" and "white," vigorous artificial respiration being recommended for the former and more passive methods for the latter. If the causes of asphyxia are examined in the light of recent work, however, it will be seen that "white" asphyxia is only a severer degree of "blue," and that vigorous artificial respiration is always contra-indicated since, far from aiding the infant to breathe, it may very well convert "blue" asphyxia into "white." The position may be summarised as follows —

After birth, if respiration does not commence efficiently at once, there will be a fall of blood-oxygen and the baby will become rapidly cyanosed (blue) unless the heart beat is already so weak that the superficial tissues are not being well supplied with blood, when the baby will appear white and limp.

*Several factors must be borne in mind when considering therapy*

(1) Prolonged anoxæmia (a) depresses the respiratory centre,  
(b) weakens the heart muscle

(2) The brain dies before the heart

(3) In the new-born baby, who has never breathed, the lungs are not expanded.

Hence methods of artificial respiration which involve pressing in the baby's chest wall are contra-indicated as they cannot help the lungs to expand and will only further embarrass the heart whose failure will lead to further anoxæmia of the respiratory centre in the brain and eventually to its death when no further efforts at resuscitation can succeed. Many babies with blue asphyxia treated with old-fashioned artificial respiration recover no doubt but the modern view is that they will recover in any case if treated more gently.

*Asphyxia neonatorum may be caused by* (a) Blockage of the respiratory passages (mouth larynx trachea bronchi or alveoli) with amniotic fluid mucus or meconium

(b) Depression of the respiratory centre by the administration of narcotics (morphia or chloroform) to the mother during labour

(c) Cerebral damage associated with cranial injury or cedema of the brain following prolonged or difficult labour

(d) A combination of the above causes

**Diagnosis and Treatment** *The following summary may be followed with advantage* (1) If the baby is blue and shows resistance to the passage of the little finger into the pharynx and the heart beat is strong failure to breathe is due in the majority of cases to obstruction in the mouth or larynx. In these cases all that is necessary when the cord has been cut is to place the baby in a hot bath and clean out the mouth and pharynx by suction. An interrupted catheter is usually adequate for this purpose though in maternity hospitals negative pressure should be on tap in the labour ward for use in these emergencies. In the majority of cases this is all that is required and the baby will commence to breathe at first spasmodically then regularly in a few minutes and will quickly gain his normal colour.

(2) If in spite of these measures respiration remains spasmodic and irregular or if the baby takes a few normal breaths and then stops depression of the cerebral respiratory centre (due to narcotism) should be suspected and a respiratory stimulant given at once. For this purpose coramine is the best drug. It is a powerful respiratory stimulant and does not appear to depress the heart. A dose of 0.25–0.5 c.c. can be given intramuscularly and repeated every six hours if necessary to the new-born infant. In extreme cases of asphyxia Moncrieff recommends giving alpha lobeline gr  $\frac{1}{2}$  which he

uncommonly in narcotised babies respiration later becomes shallow and irregular. Some infants who have undergone no birth trauma or narcotism appear to have poorly developed respiratory centres at birth and show similar symptoms. Some form of artificial respiratory apparatus is invaluable in maintaining and stimulating respiration in these cases.

Finally all babies born in asphyxia need special after care. Too often after resuscitation they are sent down to the puerperal wards in maternity hospitals without any special arrangements being made to receive them. All these babies need special care and observation. They are particularly apt to lose heat rapidly, atelectasis is a common complication and recurrence of respiratory distress is not uncommon. Each case is a different problem *e.g.* one will require repeated injections of coramine to stimulate shallow or irregular respiration, another who has undergone considerable birth trauma will need treatment for cerebral œdema (see below). Therefore it is impossible to lay down general rules for their after care except to state that every infant who has suffered from asphyxia neonatorum however slight needs continuous observation for the next few days.

**Nasal Obstruction in the New-born.** In any discussion on the causes of asphyxia neonatorum it is necessary to mention nasal obstruction. The natural instinct to breathe through the nose is so strong in the new born baby that if nasal obstruction be present death may occur through failure to establish mouth breathing. There are a large number of cases of nasal obstruction in the new born to be found in the literature (see p 419) and recently the subject has been receiving considerable attention. It appears that respiration occurs reflexly through the nose for the first ten days of life approximately. After this time the child gradually learns to use the mouth as well. If neonatal nasal obstruction is due to bony or membranous occlusion of the posterior nares respiratory failure may develop. It is probable that many of these cases have been labelled asphyxia neonatorum in the past. Most of those described in the literature were brought to the doctor during the first twenty-four hours of life for difficulty in breathing. In cases where the nasal obstruction was complete the mouth had to be held open or breathing tended to cease.

Minor degrees of nasal obstruction due to blockage of the nasal passages with mucus or amniotic fluid no doubt com

monly occur and lead to respiratory difficulty immediately after birth. Infection (colds) occurring during the first ten days of life will lead to swelling of the mucous membrane and nasal respiration may be impeded. If this occurs before the baby has learned mouth breathing respiratory distress may occur.

It is important therefore for the obstetrician to be on the look out for this condition and to have at hand a soft rubber catheter with which to remove débris from nose at birth. If occlusion of the posterior nares is present the catheter will come up against the obstruction and its failure to pass back into the pharynx will give the diagnosis. In these cases the mouth will have to be kept open suckling will be impossible and the baby will have to be fed with a spoon or by gavage till such time as mouth breathing is established and removal of the obstruction can be attempted. In treating respiratory distress during the first ten days of life partial nasal obstruction due to swelling of the nasal mucous membrane must always be borne in mind.

### ATELECTASIS NEONATORUM

Atelectasis may be defined as failure of the lungs, or part of them, to expand after birth. The condition tends to be seen in —

- (a) weakly and premature infants
- (b) in those whose respiratory centre has been depressed by prolonged or difficult labour or by narcotics
- (c) in cases where the respiratory passages have been blocked by mucus or amniotic fluid

The area affected is usually the lower lobes, particularly their para vertebral portions.

The Diagnosis is by no means easy, as it is difficult to describe any constant physical signs as pathognomonic of the condition. Atelectasis should always be suspected in cases of recurrent cyanosis, feeble crying and sucking in of the lower intercostal spaces and skin below the costal margin. When any degree of atelectasis is present a radiograph will help to clinch the diagnosis. When atelectasis is due to blocking of the respiratory passages with amniotic fluid or mucus moist sounds will be heard on auscultation. When due to feebleness, lack of breath sounds is the rule.

**The Treatment Depends on the Cause** (1) If the condition is due to blocking of the respiratory passages direct aspiration by suction up a catheter which has been passed into the trachea is indicated. Inversion of the infant is advised by Pritchard as an easier method of clearing the respiratory passages.

(2) If due to weakness the treatment consists in building up the child's strength in every way possible. The administration of thyroid as given for prematurity (see p. 33) is sometimes a help in these cases. These measures are often sufficient and the child will recover spontaneously as he grows stronger.

(3) If due to damage to the respiratory centre by intracranial pressure the treatment recommended is the same as for cerebral oedema at birth e.g. hypertonic rectal saline and if necessary cisterna puncture. Artificial respiration though advocated by some is contra-indicated.

(4) If due to narcotism stimulants should be given—coramine 0.5 c.c. six hourly and coffee salines (see p. 37).

*Pneumonia* is the most dreaded complication of atelectasis. Babies whose lungs have not expanded completely are very prone to develop broncho pneumonia if exposed to any respiratory infection. When broncho pneumonia occurs in a case of atelectasis of any degree of severity the outlook is very grave. The temperature rises and swings between 100° to 103° F. the child rapidly becomes cyanosed and may die from syncope often within forty eight hours. Hence too much stress cannot be laid on the importance of isolating nurses and relatives with upper respiratory infections.

**Treatment** Once pneumonia has set in there is little chance of curing the infant save in exceptional cases. Continuous nasal oxygen (see p. 206) is invaluable to prevent anoxæmia and cardiac failure. Coramine is the best stimulant and can be given in 0.5 c.c. doses twice a day. Half a teaspoonful of brandy given occasionally will often help the child to sleep. Finally good nursing is essential for babies at this age require constant and expert attention.

## INJURY AT BIRTH

### *The Brain*

During birth the baby is forced through a narrow curved canal and inevitably some degree of compression of the head results. It is probable that in many cases slight intracranial

damage must occur. Routine lumbar punctures has been done on new born infants, and it has been found that within the first twenty four hours 10 to 15 per cent of cases show fresh blood in the cerebrospinal fluid. From this normal condition of slight trauma to conditions of severe injury caused by prolonged labour and extraction with forceps there is an imperceptible gradation of damage to the infantile skull and brain. It is well recognised also that premature delivery and precipitate birth are just as provocative of injuries to the brain as tedious and difficult labour. The most serious intracranial birth injuries are those involving tears in the large dural sinuses, particularly those of the tentorium. On occasion the great vein of Galen may be torn across and a basal hæmorrhage result. The other factor, especially in connection with extraction with forceps, is the over riding of the parietal bones leading to rupture of the vessels entering the superior longitudinal sinus. This may produce a large hæmatoma over one or both sides of the cerebral cortex. Extraction of the after coming head in breech presentation has its own possibilities of damage to the brain. This is usually in the form of multiple petechial hæmorrhages. Altogether damage to the infant's brain during birth may be in the form of sub dural sub arachnoid and intra cerebral hæmorrhage. The bleeding may be slight and produce no clinical symptoms or it may be overwhelming in character and produce death in the first few hours of post natal existence.

In very severe intracranial hæmorrhage the child may be stillborn. In less severe cases the signs of white asphyxia may be present due to medullary paralysis, and death may ensue very shortly, though if some degree of recovery takes place the child may survive. Cyanosis, slow pulse and irregular breathing may be present and frequent convulsive seizures are the rule. The fontanelles are bulging, pulsation is absent, and the cerebrospinal fluid taken by lumbar puncture is blood stained and under pressure. Finally, when puncture is made in the lateral angle of the anterior fontanelle, free blood is obtained, and in certain cases relief of pressure ensues.

The treatment of cases of intracranial damage following birth resolves itself into four procedures. The first is the administration of hypertonic rectal saline solutions. About 1 oz of a 10 per cent sodium chloride solution is injected into the

rectum, and the buttocks are compressed for as long as five minutes if possible. This may be repeated every three to four hours. The *second procedure is to perform lumbar, cisterna, or fontanelle punctures* and so withdraw the free blood from the sub arachnoid space. In a small baby cisterna and fontanelle punctures are easier to perform and actually produce less damage than the ordinary lumbar puncture performed on the adult. Enough blood stained fluid should be taken off to allow the fontanelle to return to normal tension, to reduce the frequency of convulsions and to alleviate the cyanosis. The *third point is to keep the baby alive by proper feeding*. Too frequently these damaged children refuse to suck or swallow, and accordingly *gavage* or feeding with a stomach tube must be resorted to. The infant must be under continuous observation, his temperature and pulse carefully watched and he must be kept as quiet as possible. The fourth and last procedure, craniotomy, is one which can be resorted to very seldom and with the least degree of success. *Craniotomy in infants for cases of brain injury and intracranial or extracranial hæmorrhage is a forlorn hope*. When the management described above fails, and surgical intervention is considered it is probable that it will fail for the simple reason that too large a vessel or venous sinus is torn to be closed by any ordinary methods of stopping hæmorrhage. In the final analysis the majority of cases of severe injuries to the brain of infants during birth are fatal. The tragic feature is that in those that survive idiocy, paralysis, epilepsy, and hydrocephalus are often apt to appear later.

### Injury to Peripheral Nerves

The facial nerve may be damaged during delivery by pressure of the obstetrical forceps. Commonly the damage is unilateral and the degree of recovery depends largely on the severity of damage. The majority of cases recover after six weeks to three months but in a certain small percentage facial deformity is permanent. Treatment consists in massage of the facial muscles and galvanic stimulation. If the paralysis proves permanent, operation and anastomosis of the nerve with another nerve can be undertaken.

The brachial plexus is often injured during the course of delivery with serious results in after life. Commonly this occurs in breech presentations and extraction of the after coming arm.

There are two types of injury one involving the upper portion of the plexus the other the lower. The first type is called the *Erb Duchenne*. In this the fifth and sixth cervical roots are torn and as a result the deltoid biceps supinator longus



FIG. 7.—Baby four days old showing facial paralysis from birth injury.

infra and supra spinati teres minor and brachialis anticus are paralysed.

Paralysis of such a group of muscles produces a characteristic deformity. The hand is pronated with the forearm and the elbow is pulled in to the side. Abduction and supination of the arm become impossible and the arm hangs limply extended. Some degree of recovery is the rule but it takes six months to a year to know how great a degree of restitution will take place. During this time massage passive movements and a splint to supinate and elevate the arm are essential for contractures occur very readily. If considerable deformity is found at one year of age it is likely to be permanent. Too much traction put on the head while delivering the anterior shoulder may be another factor in producing this form of paralysis. The second form of injury is that which involves the eighth cervical and first dorsal roots and is known as *Klumpke's paralysis*. The deformity here is very different in that it involves the pronator

*muscles of the forearm and the small muscles of the hand* As a result the forearm is flexed and supinated and the muscles of the hand are paralysed The treatment consists in putting the arm up in pronation and in keeping the fingers extended Physiotherapy as described above should be persevered with for years if necessary

### Injury to Spinal Cord

This is a very rare contingency but one which must be reckoned with especially in breech presentation Traction and hyper extension of the spinal column may tear the spinal cord with or without meningeal damage bleeding and dislocation of vertebræ The immediate result is a paralysis of both lower extremities flaccid in character with complete loss of sensation and paralysis of the sphincters Recovery as a rule is incomplete and the prognosis is grave for restitution of function below the site of the trauma The possibility of survival depends largely on the height of the lesion Special apparatus plastic tenotomies and exercises may help in certain cases but a complete transection of the cord leads to death sooner or later

### Depressed Fractures of the Skull Bones

Deformities or indentations of the skull which occur during instrumental delivery as a rule pass away in a few days Depressed fractures may result and are more serious The diagnosis is obvious and if allowed to persist damage to the brain may produce permanent lesions of the central nervous system and mental defects

A simple method of restoring a depressed fracture is to compress the child's head in a direction opposite to that in which the long axis of the fracture lies and in this way the fracture may be forced out If this method fails the following procedures can be adopted the sharp point of one blade of a bullet forceps is bored through the bone at the centre of the fracture and so turned that its concavity looks uppermost Traction is applied and the depressed fracture is pulled steadily into position

In older children when the bone has become hardened and the point of the bullet forceps cannot be introduced a tiny

incision is made over the centre of the depression down to the bone. The skull is then penetrated by a gimlet, and a blunt aneurysm needle passed in and turned in a similar manner to the bullet forceps. Steady traction will reduce the depression. The skin wound is closed with one suture.

### Other Birth Fractures

The bones of the new born withstand great bending and twisting without serious injuries and when fractures occur they are usually the result of forcible traction.

The commonest fractures are those of the clavicle, humerus and femur. These fractures unite well and are only troublesome from the difficulty in immobilising a new born infant. The growth of bone rapidly corrects any deformity which may result from imperfect fixation.

*Birth fracture of the clavicle* may not be noticed as the symptoms may be extremely indefinite. Mistakes are likely to be avoided if the possibility is constantly borne in mind. The diagnosis is made by recognising an irregularity of the shaft of the bone, compared with the opposite side, and in doubtful cases a radiograph will be decisive. This fracture is treated by placing a thin sheet of cotton wool which has been powdered, in the axilla on the site of injury, and fixing the arm to the side of the chest by means of a broad bandage or binder. Union with excessive callus occurs in about three weeks, the bandage can then be removed and the child allowed to use the arm freely. The excess callus disappears within four months.

*Birth fracture of the humerus* is treated by placing narrow strips of cardboard, protected with cotton wool, around the upper arm, and fixing with a muslin bandage. The affected limb is then fixed to the side of the body with a broad bandage, with the forearm flexed across the abdomen. Bony union occurs in about three weeks.

*Birth fracture of the femur* is not uncommon, it is remarkable how little distress is caused to the child. It can be treated by extending the knee, flexing the hip, and bandaging the whole limb on to the child's body, so that the toes lie over the shoulder. Bony union takes place in about three weeks, with excessive callus formation. The bandage can then be removed, and the child allowed to kick freely. The excess callus is absorbed in about six months.

### Ruptured Blood Viscus

Rupture of the liver and spleen are the chief sources of peritoneal hæmorrhage. They may occur in breech presentations where the blood is squeezed back into the upper abdomen manipulations during version or in too vigorous resuscitations. An extensive rupture of the liver or spleen is an extremely grave accident as the bleeding is beyond control. If the capsule of the liver and spleen remains intact the hæmorrhage becomes localised for some time until the tension rises sufficiently to burst through and flood the abdomen. In the latter group of cases the child appears to be quite normal for a few days and then suddenly collapses. The abdominal symptoms are vague and the signs indistinct. The hæmoglobin index tends to be low.

**Treatment** If one is fortunate enough to see a case of hæmoperitoneum sufficiently early after collapse an immediate laparotomy should be done and the liver exposed. Packing with a length of gauze the end of the gauze being left protruding from the abdominal wound is generally the only procedure to be adopted. This packing is left *in situ* for four or five days and then gradually removed.

### Ruptured Bowel

Is an uncommon occurrence in the recorded cases the large intestine is more often involved than the small intestine and this may be accounted for by the fact that the colon is often distended with meconium late in gestation. The clinical features are those of peritonitis the child refuses to suckle continuous crying due to pain vomiting the abdomen becomes tympanitic and distended. A radiograph may show distended loops of intestine with fluid levels due to intestinal obstruction from the peritonitis. There is a leucocytosis.

**Treatment** Immediate laparotomy should be done under local anæsthesia.

## CHAPTER VII

W R F COLLIS

### COMMON DISORDERS OF THE NEW-BORN

(Jaundice (Classification) Icterus Gravis Signs Symptoms and Treatment Hæmorrhage (Classification) Hæmorrhagic Disease of the New born—Acute Infections—Dehydration Fever—Œdema of the New born Scleredema—Sclerema—Mastitis—Stomatitis—Constipation—Tongue Tie )

#### Jaundice

##### Classification of the Causes of Jaundice during the Neo natal Period

- A ANATOMICAL—congenital obliteration of the bile ducts
- B Physiological—slight jaundice occurring during the first week
- C Hæmolytic—Icterus Gravis Neonatorum
- D Infective—(1) *Syphilis* (2) *acute hepatitis due to pyogenic infection from umbilicus*

Complete obliteration of the bile ducts is a rare form of congenital malformation the outlook is hopeless and the child gradually becomes more and more jaundiced until death supervenes. Certain cases linger on for considerable periods of time, one case has been reported recently that lived until six months of age.

Physiological jaundice occurs in some 30 per cent of normal babies. The average baby is born with some 6 000 000 red blood corpuscles per cubic millimetre. While the baby is still *in utero* the oxygen tension in his blood is lower than after birth and hence a number of the unwanted red cells are broken down immediately the infant begins to breathe. In those cases where the child is born with an unusually large number of red blood corpuscles or when the hæmolysis after birth is particularly rapid a degree of jaundice visible to the naked eye, appears for a day or so. It is only of importance in so far as it may be confused with the graver forms of jaundice which may occur at this period.

**Icterus Gravis Neonatorum** Under this heading a number of different conditions are described. If as sometimes occurs the normal hæmolytic just described is carried too far and more than the correct number of red blood corpuscles are destroyed a deep jaundice appears which is later followed by anæmia. The condition can usually be checked easily by the injection of 10 c.c. of the mother's blood into the infant if diagnosed early enough and a transfusion will always remedy the situation if the anæmia produced is more than transitory. This form of jaundice is commonly met with in premature infants whose red blood corpuscles are more fragile than normal. Some obstetricians guard against its occurrence by the routine injection of 10 c.c. of maternal blood into every infant at birth. True icterus gravis is however quite a different condition. It has a definite family history, syndrome of symptoms, course, blood picture, treatment and post mortem appearances. These may be summarised as follows.

*Family History* The condition will suddenly appear in a family. One or two normal babies may be born and reared satisfactorily, then for no apparent reason a baby is born and dies (as a rule) within a week or ten days of birth, the chief symptom being a very profound jaundice. After this every child the mother bears behaves in the same way. The condition is not directly hereditary but there is some evidence that a Mendelian recessive factor may play a part.

*Symptoms* Shortly after birth, sometimes within six hours, the jaundice commences and rapidly increases in intensity until the child becomes a deep copper colour. Later as the anæmia proceeds the colour will lighten and the child assume a very characteristic yellow wax pallor. The liver and spleen rapidly become enlarged, the child loses weight and becomes drowsy and disinclined to take nourishment. Death may supervene within five days to three weeks if the condition is left untreated.

*Blood Picture* This is highly characteristic. The picture presented is that of a severe hæmolytic anæmia in which an acute destruction of red cells is proceeding rapidly while at the same time all the hæmopoietic tissue in the body is pouring out new cells to take their place but failing to do so rapidly enough. Hence we find large numbers of normoblasts (I have counted as many as 200 000 nucleated red blood corpuscles per cubic millimetre in a film from a case in the Rotunda Infants Clinic).

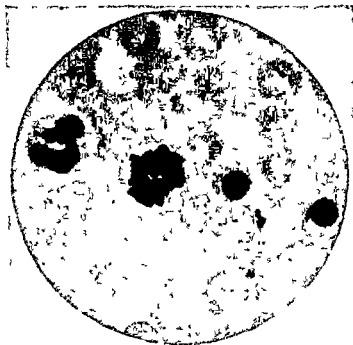


FIG 8.—Blood Film  $\times 1300$  (from left to right) polynuclear leucocyte, megakaryocyte, mitosis, and two normal leucocytes.

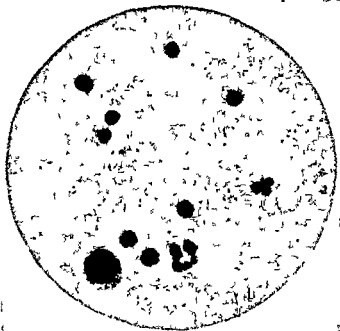


FIG 9.—Blood Film  $\times 800$  showing numerous normal leucocytes.

and reticulocytes. The cells appear of all shapes and sizes, and polychromasia may be present. There appears also to be a great activity in white cell formation. Large numbers of myeloblasts and myelocytes may be seen in the field. In fact, blood films from these cases contain every form of blood cell and are highly interesting to hæmatologists (see Figs 8 and 9). The number of red cells decreases rapidly, and in an untreated case may be below 2 500 000 per cubic millimetre within forty eight hours of birth. The colour index is always above 1.

As the disease progresses the red cells become fewer and fewer in number, the count sometimes falling below 1,000 000 per cubic millimetre. The response of the reticulo endothelium gradually diminishes and fewer immature forms are found as the case nears the fatal ending.

Cases coming to post mortem show very characteristic appearances, the most prominent feature being extra medullary hæmatopoiesis. This is found in the liver and spleen chiefly, but is also sometimes seen in the pancreas, adrenals, gonads, intestinal tract, lymphatic glands, connective tissue, skin and other tissues.

*The liver* is enlarged, shows many areas of hæmatopoiesis, deposits of bile pigment and hemosiderin. The degree of degeneration or fibrosis will depend upon the date of death.

*The spleen* is enlarged and shows similar areas of hæmatopoiesis.

*The bone marrow* is hyperplastic and shows a condition of brisk hæmatopoiesis.

*The heart* sometimes shows hypertrophy.

*The Nervous System.* Here the changes may be connected with (a) hæmorrhage, (b) icteric staining followed by degeneration (kernicterus).

Icteric staining may occur in the choroid or meninges or in the basal ganglia of the brain substance itself. The latter may be followed by various neurological phenomena associated with degeneration of the basal ganglia.

*Treatment.* Different treatments have been suggested and claimed to be satisfactory, e.g. liver extract, adult serum, blood transfusion. In our experience, when dealing with a genuine case of the disease, blood transfusion has been the only curative procedure. Recently campalon injections combined with transfusion have appeared to accelerate recovery. Direct

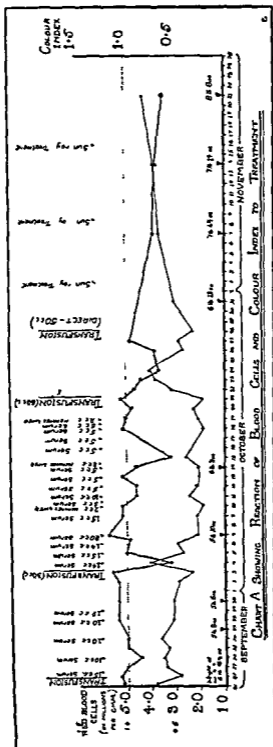


FIG 10

transfusion has appeared more valuable in our cases than the citrate method (indirect)

The diagram on p 55 illustrating the treatment given to a case in the Rotunda Infants Clinic is illustrative of these points. If it is studied it will be seen that the number of red blood corpuscles had fallen below 4 000 000 per cubic centimetre when the first count was done (forty-eight hours after birth) the colour index being above 1. A transfusion was performed at once but was followed by only temporary improvement. A week later a second transfusion was performed and ten days later a third. Each was followed by a temporary improvement only to be superseded by another attack of hæmolysis and it was not till after the fourth transfusion (this time by the direct method) that recovery took place.

It will be seen that human serum and liver extract were also given but without apparent beneficial effect.

**Diagnosis.** As can easily be seen from the above description it is difficult to diagnose the condition in most of these cases early enough to save the child's life except when the mother gives a history of having already lost one or two babies with acute jaundice. Treatment to be effective must commence within twenty-four hours of birth. Hence except when the patient is in a hospital where there is a pathological department able to carry out a complete blood examination at a moment's notice there is little chance of successful treatment. The majority of cases are diagnosed too late if at all. The author regards the history as of paramount importance.

Doctors and nurses should always be on the look out for such family histories and if they receive them preparations to deal with the situation should be made before the baby is born.

**After Effects.** If the treatment is prompt and the case is not very severe complete recovery is probable. If treatment is delayed or if the case is particularly severe certain after effects may occur. The liver may have been severely damaged and cirrhosis may follow. Certain areas in the central nervous system may be picked out and damaged by the disease. The commonest area is the lenticular nucleus which may be irreparably damaged during the acute illness and later gradually degenerate. This condition of liver cirrhosis and lenticular degeneration is very like the neurological syndrome described

by Wilson in adults, and icterus gravis neonatorum may indeed prove to be the precursor of Wilson's disease

The cause of the condition is unknown. We may postulate that normally there is some hæmolytic factor present in all bloods which is balanced by similar anti hæmolytic substance, their interaction keeping the blood at a normal level throughout life. In the case of icterus gravis this anti hæmolytic substance would appear to be absent. The fact that transfusion cures the condition would suggest that the anti hæmolytic substance is supplied in the donor's blood.

Two other conditions, hydrops foetalis and anæmia gravis neonatorum (without jaundice), are now considered to be different clinical manifestations of the same underlying condition as icterus gravis, and all three have been grouped by American writers under the single title *erythroblastosis foetalis*. The whole subject is still under investigation and cannot be discussed further in a work of this size.

**Infective Jaundice Neonatorum.** A *Syphilitic jaundice* may commence during the first week of life and rapidly prove fatal. The prognosis depends entirely on the degree of liver damage and the promptness of the treatment. In severe cases where the liver contains many spirochætes death occurs early. In milder cases where treatment has been given from birth, recovery occasionally takes place.

**B. Hepatitis due to infection of the cord** by pyogenic organisms spreading up the umbilical vein, is a very serious complication of the neo natal period. It is associated with high fever and the outlook is very grave, death taking place as a rule in a few days.

Cases of *catarrhal jaundice* have been reported during the first week, but there is little real evidence that true catarrhal jaundice ever occurs at this age.

Cases of *acholuric family jaundice* have been reported during the second week, but they are merely medical curiosities.

### Causes of Anæmia Neonatorum

**Hæmorrhage.** (a) Bleeding may occur from birth injuries, particularly lacerations.

(b) *Umbilical bleeding* may be of several varieties; it may be due to faulty tying of the cord at birth, injury, sepsis or hæmorrhagic disease of the new born. The treatment is symptomatic.

(c) *Vaginal hæmorrhage* During the first week baby girls not uncommonly have a mucous vaginal discharge and some times this is mixed with blood from the uterus. It clears up spontaneously as a rule. very occasionally it may be severe when associated with hæmorrhagic disease of the new born.

(d) *Hæmophilia*

(e) *Hæmorrhagic Disease of the New born* This is a comparatively common form of hæmorrhage in infants. Its cause is not altogether understood but is thought to be associated with a failure on the part of the body to form prothrombin, which defect in the infant is considered by some workers to be due to a vitamin B deficiency in the mother during pregnancy. It affects boys and girls equally and occurs usually about the second day of life. The bleeding may be profuse and if unchecked may endanger life. Melæna is the usual form in which the hæmorrhage occurs though it may take place from the umbilical cord, vagina, kidneys, skin or mucous membrane of the respiratory passages. The motion usually appears reddish black, of the consistency of tar and has a foul odour. It has to be distinguished from the normal meconium which is greenish and odourless. When the bleeding is from low down in the alimentary tract the stool may be bright red in colour. Associated with the melæna appear symptoms of restlessness, sub-normal temperature, pallor and coldness of the extremities—in short the infant appears shocked. The appearance of the hæmorrhage is often dramatic and terrifying for the mother or nurse. As treatment when applied early enough is usually successful the relatives should be reassured and calmed. *Immediately 10–20 c.c. of whole blood should be injected into the baby.* The donor can be one of the parents or any adult. The injection should be repeated in three hours whether a further hæmorrhage takes place or not. If the hæmorrhage is controlled by these measures the injection of further blood is unnecessary. But if further hæmorrhage occurs injections of blood should be continued intramuscularly till it ceases or better if the apparatus is to hand a blood transfusion should be performed without further delay. In any case if the child is left anæmic or debilitated after the attack a blood transfusion is much the best form of treatment.

When the doctor reaches the baby he usually finds that it is cold and dehydrated and severely shocked hence he should place it at once in an incubator or in a cot warmed with hot

water bottles. Even when the bleeding is alimentary it is usually advisable to continue fluids by mouth to combat the dehydration. If the latter is very severe normal saline should be given subcutaneously or intraperitoneally.

### ACUTE INFECTIONS

Infants may become infected *in utero* with certain acute infectious diseases (e.g. they may be born with measles, typhoid fever etc. or in the incubation period for one of these diseases and develop it shortly after birth).

The new born baby has very little resistance to the invasive power of micro-organisms although it may possess a passive immunity to certain diseases for the first few months of life. Hence infection with pyogenic organisms is a common occurrence, and one of considerable gravity at this age. Suppuration occurs rapidly, and the infection is always liable to spread, becoming pyæmic or septicæmic, complicated by pneumonia or meningitis. The route of infection may be through an abrasion on the skin or by way of any of the mucous membranes or by the umbilicus the usual infecting organisms being the streptococcus, staphylococcus and pneumococcus.

#### *Infections of the Umbilicus*

In spite of every care in dressing the umbilical cord may become infected by pyogenic organisms. The most serious degrees of infection may result in (1) Septic omphalitis (2) Gangrenous omphalitis (3) Infective thrombo arteritis and phlebitis of the umbilical vessels.

**Septic Omphalitis** When the cord has separated an ulcer forms at the umbilicus from which there is a sero purulent discharge and the infection may spread into the surrounding cellular tissues. The skin round the umbilicus becomes raised and red, with all the local signs of inflammation and in severe cases spreading infection occurs towards the thighs like an erysipelas.

**Treatment** The ulcerated area should be cleansed with a weak warm solution of hydrogen peroxide, and dusting powder applied. In severe cases incisions and fomentations may be necessary.

**Gangrenous Omphalitis** This condition is due to a virulent

streptococcal infection. Gangrene occurs in the tissues around the umbilicus and the prognosis is grave.

**Infective Thrombo arteritis and Phlebitis** The infection spreads in the umbilical vessels and may follow a septic omphalitis. All the signs common to septicæmia may occur. The baby becomes extremely ill, jaundice is an early and prominent symptom, hæmorrhage may occur from the stump of the cord, the pulse is rapid and the disease is invariably fatal.

Erysipelas is not uncommon, commencing either from the umbilicus or some small abrasion and spreading rapidly (see p. 176).

We have already mentioned the rapidity with which pneumonia will develop during this period in babies exposed to infection.

The new born are also particularly prone to develop impetigo called pemphigus neonatorum (see p. 316).

Treatment is chiefly prophylactic. If the measures outlined in the preceding chapters are carried out few cases of neo natal sepsis will occur. But if nurses, handy women, relatives or friends when they have septic fingers or respiratory infections are allowed near the new born, sepsis is very apt to occur.

Active treatment is of very little avail in severe cases at this age. If localised abscesses form they must be treated symptomatically by surgical measures. Transfusion is a measure of real value if the infection is localised and in erysipelas but in the severer forms it is not worth attempting.

Ophthalmia neonatorum is described fully in the ophthalmic section (see p. 309).

### Dehydration Fever

A definite clinical syndrome associated with dehydration has been described in the new born. During the first few days of life before the breast milk comes many babies are not given additional fluid. Hence fluid loss from the body by way of the skin, kidneys and alimentary canal may very greatly exceed that of the intake. This is particularly apt to occur in hot weather or if the baby is placed in an incubator. In these cases the temperature may rise rapidly to 102°-104° F. while the skin loses its elasticity and lies in folds. The fontanelle is sunken, weight is lost rapidly and prostration is marked. It is easy to mistake the condition for some infective state, these

however are seldom seen before the fifth day. In such cases if no source of infection can be found dehydration fever must be assumed to be the cause and the child treated accordingly. Fortunately the treatment is simple and effective. If additional water is given by mouth together with small rectal salines the temperature usually falls at once and the child recovers. In many severe cases a subcutaneous saline should be given as soon as the condition is diagnosed. If the child is drowsy and disinclined to suck fluid should be administered by gavage. Prophylactic treatment is all important for the administration of additional fluid during the first few days will prevent the occurrence of the condition.

### Œdema of the New born

There are a number of different types of œdema (other than cerebral œdema) met with in the new born. Infants born of toxæmic mothers (e.g. eclampsia) not uncommonly show a generalised œdema. In other cases there is no apparent cause but shortly after birth a hard brawny œdema appears first on the dorsum of the feet later spreading over the body. This condition which has been called *scleredema* is usually met with in premature and weakly infants. We have found that hypertonic rectal salines are of great value particularly if given before the condition has had time to develop extensively.

Sometimes it is complicated by sclerema.

### Sclerema

This condition is essentially a solidification of the subcutaneous fat either in circumscribed areas or throughout the body. Normally fat is in a semi liquid condition in these cases it becomes solid as in the cadaver. The cause of the condition is not fully known. The melting point of fat in the infant is higher than in the older child and hence it is supposed that a fall in skin temperature may lead to sudden solidification of the infant's subcutaneous fat though no doubt other factors also play a part. It is usually seen in feeble infants during the first week but it is occasionally met with later and may be associated with marasmus as late as the third month.

The baby has a characteristic appearance the skin first of the calves and later in patches all over the body becomes indurated and hard to touch but does not pit on pressure.

Over the buttocks it often appears lobulated The colour of the baby is bluish, or sometimes slightly yellow The extremities are stiff and move with difficulty The child has a feeble cry, slow and feeble respirations and lies in the cot resembling a dead rather than a living baby The temperature is usually very much below normal, often between  $86^{\circ}$ – $90^{\circ}$  F

The prognosis is always bad, the majority of cases ending fatally

Treatment consists in heating up the baby by every available means—externally with electric blankets incubators hot water bottles etc internally by stimulating metabolism by the administration of thyroid (see p 33) If the condition is associated with dehydration subcutaneous saline infusion may be given

Urinary disorders in the new born such as anuria, pyelitis, etc are dealt with elsewhere (see p 244)

## CERTAIN MINOR DISORDERS OF THE NEO-NATAL PERIOD

### Mastitis

Lactation associated with hyperplasia of the infant's mammary tissue is a very common occurrence in the new born There is often quite a copious secretion of milk analogous in every way to mother's milk The condition is due to the passage of maternal hormone through the placenta into the circulation of the foetus The condition occurs in both boys and girls As a rule if left alone, it clears up without complications If the baby's swollen breasts are squeezed rubbed or allowed to get dirty infection may take place and a mild or severe mastitis supervene depending upon the infecting organism In most cases hot poulticing will reduce the inflammation but occasionally suppuration occurs and surgical drainage becomes necessary

The condition is somewhat surprising and alarming when seen for the first time hence the doctor must reassure the mother, explain to her that it is nothing abnormal or pathological and impress upon her the importance of leaving it alone

### Stomatitis

Inflammation of the gums mucous membrane of the mouth and tongue is not uncommonly met with in the neo natal

period and during the first few months of life. It is due to dirty feeding—in the breast fed baby to failure on the part of the mother to keep the nipples clean, in bottle fed babies to dirty teats. It is particularly likely to occur in diseased and weakly infants.

Several varieties are described —

### (a) Thrush

Here the mucous membrane is invaded by a parasite (*Oidium albicans*). At first it appears as a white patch on the tongue or mucous membranes. Later these patches coalesce until possibly the whole buccal mucous lining is thickly coated with a greyish white scum. This false membrane may extend into the oesophagus.

Treatment consists of (1) improving the child's general condition (2) Giving instruction as to correct feeding (3) Painting the inside of the mouth with gentian violet (1 per cent) daily and cleaning out the cavity three times a day with glycerin and borax.

### (b) Aphthous Stomatitis

This is due to a localised infection of the mucous membranes. In the early stages there is a fibrinous exudate. Small spots subsequently develop which break down and become painful ulcers. The abnormality is often associated with general ill health. The best treatment is to cleanse the mouth frequently with some mild disinfectant (glycerin and borax) and apply peroxide paste directly to the ulcers.

### (c) Bednar's Aphthæ

Consist of single or dual superficial ulcers above and somewhat mesial to the tonsillar fossa on the hard palate (actually they are situated as a rule on the hamuli of the pterygoids). There is no surrounding area of inflammation. They tend to occur in badly cared for and sickly infants of poor mothers, but we have seen them even in nursing homes when feeding bottles have not been properly sterilised. They appear to be caused by trauma while cleaning the mouth or friction from a dirty rubber teat. When met with in weakly infants they are a very bad sign and may be very intractable. If the child's

general health can be improved however and if they are treated as described above for aphthous stomatitis healing may occur rapidly

### Constipation

Constipation is often troublesome in the new born baby and later during infancy

There are two main types —

(1) *Due to anal stenosis or spasm*

(2) *Due to bad management insufficient fluid etc*

In anal stenosis if the vaselined finger is introduced into the rectum and the anal sphincter stretched a moist normal motion will follow its withdrawal. If the cause be true constipation the stool will be hard and dehydrated. This may seem a small point but it is one of considerable importance. Anal stenosis is commoner than is generally supposed and it is useless giving aperients or attempting to train an infant under these circumstances. Also the condition is easily cured one or two stretchings with the vaselined finger being enough usually to cure the condition.

The treatment of the other forms of constipation depend upon their cause —

(a) **Insufficient Fluid** This is the commonest cause in the infant and can be corrected by giving additional bottles of water (at least two during the twenty four hours) to all normal babies

(b) **Lack of Proper Attention and Management** Training from the first by placing the baby at regular intervals on the chamber is of paramount importance. Sometimes the reflex of defecation can be made a habit at these times by stimulating the rectum with the vaselined finger or glycerine suppository on a number of occasions. The regular holding out of the baby should be insisted on even if it is resisted at first. If training is systematic defecation soon becomes an established habit.

(c) **Anal Fissure** is rare in the new born but fairly common in later infancy due to the tearing of the mucous membrane by a hard constipated motion. Stretching of the sphincter followed by a mild ointment will usually cure the condition though in later childhood it may be troublesome. The fissure makes the act of defecation painful and the infant resists the desire to defecate till the reflex passes off.

(d) **Overfeeding or Underfeeding in Breast-fed Babies and Irregular or Wrong Artificial Feeding** Here the remedy depends upon correcting the primary error. See section dealing with nutrition.

In a large number of infants, however, in spite of good management and correct feeding, some degree of constipation is often found and further measures will be required. Olive oil is useless as an aperient, and castor oil should never be used as a cure for constipation. It clears out the bowel and then constipates the child again. In fact, castor oil does more harm to babies than any other drug. It is not uncommon to have babies brought up to hospital who have been given castor oil once a week with the result that except for a number of motions following the weekly dose the child has become completely constipated.

The essence of the treatment of constipation is the *establishment of habit*; hence any medicine given must be administered regularly. Liquid paraffin is by far the best aperient to use. It is non-irritant and non-absorbent, it moistens the stool without griping the baby, and it may be given in any reasonable dose. Hence liquid paraffin should always be given regularly in all cases of constipation during infancy, particularly during the *neonatal period*. Milk of magnesia may also be given regularly, but should not be continued indefinitely as it tends to lose its action and may eventually lead to further constipation. Senna (made as an infusion) may be given if liquid paraffin and milk of magnesia fail, but it will seldom be necessary if management, fluid intake and diet are correct.

### Tongue Tie

There is a tradition that this condition is a common complaint in new-born infants. Actually it is doubtful if a genuine shortness of the *frænum linguæ* is ever present. Certain small or poorly developed infants appear to be unable to protrude the tongue, hence the *frænum* is not stretched and appears shorter than normal. Snipping the *frænum* is seldom, if ever, justifiable as it never cures the difficulty in sucking, which is usually the cause for the child being brought to the doctor. Hence doctors should reassure the mother, and refuse to snip the *frænum* even when begged to do so.

## CHAPTER VIII

W R F COLLIS T J LANE C L McDONOUGH

### CONGENITAL OR HYPERTROPHIC STENOSIS OF THE PYLORUS

(*Ætiology—Pathology—Diagnosis Vomiting Constipation Visible Peristalsis Palpable Tumour Reaction to Treatment Radiological Diagnosis—Duodenal Atresia—Choice of Treatment Medical, Surgical—Operative Technique—Post-operative Treatment—Summary of Treatment.*)

PYLORIC stenosis though a rare condition occurs with sufficient frequency to make it one of the most important diseases of infancy. Every doctor engaged in practice should be able to diagnose the condition in its early stages for if the baby is to be saved it must be treated without delay. Unfortunately many cases are still diagnosed too late or unrecognised altogether and left to die. Hence a chapter is devoted here to the subject and the signs symptoms and treatment of the condition briefly set forth.

The main features of the disorder may be summarised as follows: the mother gives a history of a normal delivery and of the infant being apparently quite normal at birth. The child is usually the first born and nearly always a boy. For the first two weeks he thrives then suddenly during the third week he ceases to gain and begins to vomit while the motions become irregular. Soon the vomiting becomes regular occurring after every feed or every other feed the contents of the stomach being expelled with great force. *Projectile vomiting* is characteristic of the condition. The feed is shot out through the mouth and nose sometimes to a distance beyond the infant's feet it may be streaked with blood or blood may be intimately mixed with the vomitus giving it a coffee ground appearance though this is a rare complication.

Not uncommonly when the condition has been unsuspected by the doctor the child is sent up to hospital eventually, where the history of the case shows that the feeding has been changed

four or five times in the hope of finding a "suitable mixture," but without avail, each new feed being vomited in the same way. This is a good example of the commonest fault in the management of infant feeding, *i.e.*, changing the feeding from one formula to another without a definite reason. The importance of always having a clear reason before altering the feeding of any child cannot be over emphasised, more often than not it is some condition (such as pyloric stenosis) in the child, not an unsuitable diet, which is causing the trouble.

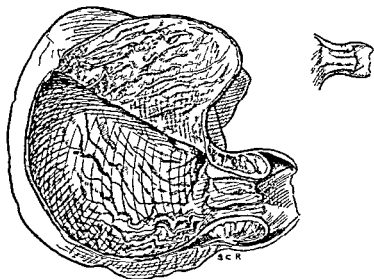


FIG 11 —Sketch of stomach from case of pyloric stenosis showing hypertrophied circular muscle of pyloric sphincter enfolded mucous membrane and much dilated stomach.  
*Inset* Normal pylorus drawn to same scale

At first the bowels are only slightly irregular but gradually constipation increases.

The child's general appearance varies with the length of time which has elapsed since the vomiting has commenced. If the diagnosis is made within some days of the onset he may be well covered and comparatively normal. If the condition has existed for some weeks, however, the baby will be much wasted and dehydrated, in long standing cases he may present a picture of extreme emaciation.

**Anatomical Pathology** The appearance of the pylorus is remarkably constant in pyloric stenosis. It appears whitish,

spindle shaped varying in length from  $1\frac{1}{2}$  to 2 inches and in breadth from  $\frac{1}{4}$  to 1 inch. The hypertrophy occurs in the circular muscle which may be many times its normal thickness. The mucous membrane is thickened also and plicated and usually œdematous. The stomach is always dilated with some degree of mucous gastritis the mucous membrane being gathered into longitudinal folds.

The pathogenesis of the condition is not as yet fully understood. One theory is that it is due to faulty sympathetic enervation of the stomach and pylorus which causes the pylorus to contract firmly instead of relaxing when the peristaltic wave reaches it from the stomach. Another theory is that the condition is due to a primary congenital hypertrophy. Undoubtedly both spasm and hypertrophy play a part in the condition. The fact that cases with hypertrophy have been found at birth and even in certain premature babies suggests that a primary hypertrophy must play a part even if spasm later aggravates the condition. There is no agreement as to the underlying cause of the whole state however and a discussion here between such different theories as a vitamin B deficiency and hyperadrenalism would in our opinion only confuse the student.

**Diagnosis.** The main features of pyloric stenosis are very constant. When a male first born baby about three weeks old is brought up complaining of vomiting of a violent type the condition should always be suspected. It must be remembered however that the condition occurs occasionally in females and in other than first born babies. Sometimes it occurs in twins or one baby after another in the same family. On one occasion a mother came up to the Potunda Hospital with a baby and said he had pyloric stenosis. He had. Her previous child had also suffered from the condition and she recognised the symptoms. Again although the third week is the commonest age for the symptoms to commence it is by no means always the rule. Out of 197 of Still's cases thirty began to vomit within the first week and three of these within the first forty eight hours. Only two of his cases however commenced to vomit later than the eighth week. These figures are similar to those of other writers including the present authors most of whom have diagnosed quite a number of cases during the first week of life but have not seen more than 1 per cent commencing after the eighth week.

The diagnosis rests on four definite features which are present in every case :—

*Vomiting.*  
*Constipation.*  
*Visible peristalsis.*  
*Palpable tumour.*

We have already dealt with the symptoms of vomiting and constipation and need now only describe the latter two cardinal signs.

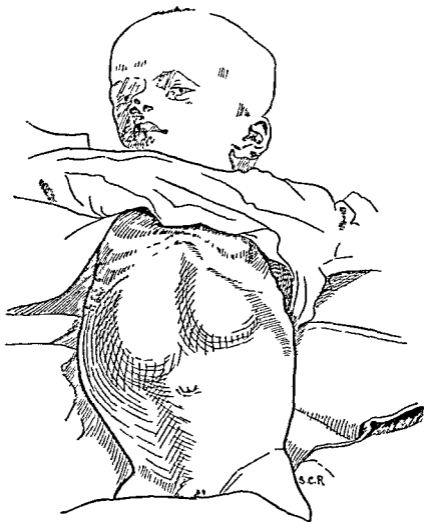


FIG 12—Sketch showing visible peristalsis in a case of pyloric stenosis.

**Visible Peristalsis** On examining the abdomen you will observe that the upper part appears full when compared with the lower part. This is due to dilatation of the stomach. If the child has vomited since its last feed this may be all that can be seen at the time. If however the stomach is full waves of peristalsis may be observed through the abdominal wall passing from left to right across the organ. They are very characteristic appearing like large moving balls. In marked cases they may elevate the abdominal wall almost an inch over their central point in slight cases they may be hard to see. To bring out the peristalsis and as we shall see to feel the tumour it is always wise to feed the baby while the examination is being made. The peristaltic wave may sometimes be stimulated by flicking the abdominal wall over the stomach with the finger or by placing a few drops of ether from a pipette on the same area.

**Palpable Tumour** Considerable disagreement is found over this question. Some workers maintain that it is often impossible to palpate the tumour others that it can be felt in every case. In the present authors experience it is largely a matter of patience. The physician must not be in a hurry. He will have to sit down beside the child while it is being fed and continue palpating for a considerable time sometimes for as long as fifteen minutes before the tumour can be felt. It is often tucked up under the liver and can only be felt when a peristaltic wave reaches the pyloric end of the stomach and pulls the hypertrophied pylorus down under the finger. Some have suggested that it is not the pylorus at all that is felt but the peristaltic wave as it reaches the pyloric end of the stomach. This explanation is not tenable however as the tumour is often felt when no peristalsis is taking place.

In our experience if the following routine is observed the tumour should be felt in almost every case. (Out of some fifty cases we have only failed to palpate the tumour in two.)

The baby is placed in the mother's left arm and given a bottle with the right hand his abdomen is uncovered. The physician sits on the baby's left side and palpates the child's abdomen with his left hand passing the middle finger in behind the right rectus muscle. The tumour will be felt like a hard hazel nut or marble deep in the abdomen somewhere between the umbilicus and the liver a little to the right of the border of the right rectus. It will not be felt all the time but will come up against the finger and then recede again.

Till one real tumour has been felt by the student he is inclined to imagine sometimes that he feels a tumour, when in reality it is not present, but once a genuine tumour has been palpated he will never make the mistake again, as the sensation it gives to the finger is very characteristic

Again we must emphasise the point that the tumour may not be felt at once and that the examination may last for from fifteen to twenty minutes before it is successful, or may even have to be repeated before the tumour is felt

In our opinion, however, this sign is of cardinal importance, and the greatest patience should be exercised in eliciting it. It is the only sign which is absolutely diagnostic of the condition. If the tumour is felt the diagnosis is certain, if not it is open to doubt. Pylorospasm may simulate pyloric stenosis in all but the last sign. Pylorospasm has been called the gastro enterospasm of hypertonic infants. It is associated with very similar signs and symptoms to pyloric stenosis. There is progressive loss of weight, forcible vomiting, visible peristalsis and constipation. It may occur in babies older than those liable to suffer from pyloric stenosis but is not uncommonly found during the first six weeks of life. The diagnosis rests, as we have said, primarily on the question of the tumour. In any case where, after prolonged and careful palpation by an expert, the tumour has not been felt the diagnosis of pylorospasm must be considered carefully. Further help can be obtained —

(1) By observing the child's reaction to treatment

(2) By giving a barium meal and observing its passage on the screen or by taking a number of radiographs

**Reaction to Treatment** Give atropine  $\text{M}_{1-11}$  of a 1000 solution of atropine sulphate with each feed increase by  $\text{M}_1$  gradually till an atropine flush occurs. Then reduce the dose till just below this amount. In cases of pylorospasm the condition is relieved by this means while it has little effect on a case of pyloric stenosis. Atropine treatment has certain dangers in the small infant. Atropine poisoning may occur with fever, rapid pulse, flush and sometimes collapse. Recently the Scandinavians have introduced eumydrin (atropine-methylurate) and claim considerable success with its use. The method of administration is as follows. 5 c.c. of a  $\frac{1}{1000}$  solution (i.e., 0.5 mg.) are given half an hour before every feed (e.g., seven times in the twenty four hours).

Luminal if given in sufficiently large doses (gr  $\frac{1}{10}$ — $\frac{1}{8}$ ) to produce drowsiness in the infant is claimed also to be of considerable value in cases of pylorospasm

### Radiological Findings in Congenital Pyloric Stenosis and Pre pyloric Spasm

The normal stomach in infancy lies almost horizontally and at a higher level than in the adult even when examined in the upright position and comparatively little alteration in shape occurs with change of posture. A disproportionate amount of air in the stomach of the infant is a constant radiological finding.

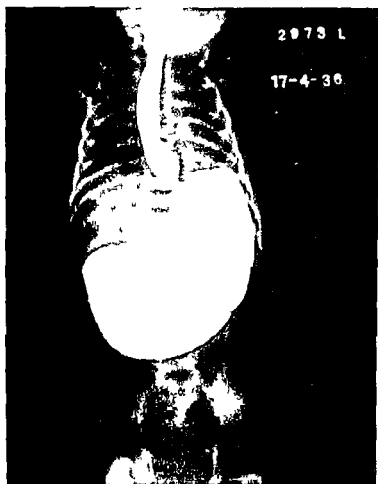
The average infant stomach will evacuate 3 oz barium cream within one to two hours and soon after ingestion the pylorus and duodenum are clearly outlined. Complete retention of barium in the stomach for a period exceeding two hours denotes in the great majority of cases hypertrophy of the pyloric sphincter and such observation should be followed by further examination at intervals up to twelve hours. If at this period no barium has passed the pylorus a diagnosis of hypertrophic stenosis may be made with confidence.

A group of patients with some of the clinical signs of pyloric obstruction (*e.g.* visible peristalsis, constipation and projectile vomiting) prove on radiological examination to be suffering from spasm, practically always affecting the pre pyloric segment. In such cases a part of the barium usually escapes into the duodenum within a few minutes and spasm then develops preventing further egress for thirty to sixty minutes but very seldom for a longer period.

The appearance of the stomach in such cases is somewhat similar to that following partial gastrectomy, the distal portion being quite free from opaque material since the lumen is occluded by the powerful contraction of the walls. In the milder cases of spasm the outline of the pre pyloric segment is still visible but owing to the diminished lumen the mucous folds are more numerous and prominent.

There are intermediate grades of hypertrophic stenosis which range from a slight narrowing of the pyloric canal to the reduction of its lumen to a mere capillary passage difficult to demonstrate. In the latter case the amount of opaque material reaching the small intestine is often insufficient to give a

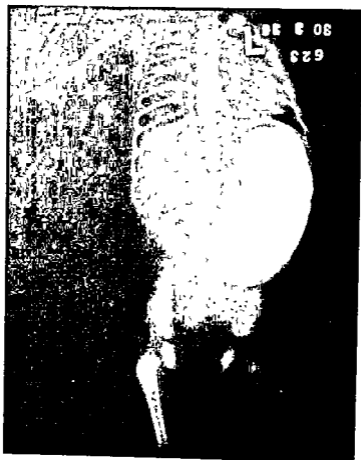
PLATE I



CASE 1 —PYLORIC STENOSIS

No emptying, one hour after barium meal. Reflux into esophagus

PLATE II.



CASE 2A —PYLORIC STENOSIS

Eighty minutes after barium meal. Fairly active peristalsis. Small amount of barium in first stage of duodenum, indicating pyloric canal of capillary dimensions.

definite shadow and for this reason a preliminary radiograph should be made as a routine immediately before the administration of barium. This will often allow one to decide whether or not obstruction is complete.

To sum up

The normal infant stomach empties within two hours.

Complete retention of barium for two hours indicates hypertrophic pyloric stenosis in the great majority of cases and if retention persists up to twelve hours the diagnosis is certain.

Initial evacuation of a small amount of barium followed by a period of retention which seldom exceeds one hour and subsequent resumption of emptying indicates pyloric or more commonly pre pyloric spasm.

Intermediate grades of pyloric stenosis may be encountered from almost normal emptying to the practically indistinguishable thread like passage. In these cases the amount of barium and the regularity of its distribution in the small intestine give one a guide to the degree of stenosis even when difficulty arises in visualising the pyloric canal (see Plates I II III and IV).

**Duodenal Atresia** The only other condition which may be confused with congenital pyloric stenosis is congenital duodenal atresia. This very rare condition is characterised by the onset of vomiting occurring in the first twenty four to forty eight hours of life by the vomitus usually containing bile and by the absence of a tumour. In a case recently diagnosed before death in the Rotunda Hospital an x ray examination showed a dilated stomach full of barium the duodenal cap and the first part of the duodenum and then complete stoppage. Laparotomy was performed with a view to attempting gastro-enterostomy but the bowel below the stricture was found to be atretic and nothing could be done.

No other form of abdominal condition should lead to confusion in the diagnosis. Acute gastritis is occasionally diagnosed as pyloric stenosis when the vomiting is persistent but only by those who are unfamiliar with the perfectly definite syndrome presented by pyloric stenosis. In gastritis the vomiting is not characteristic the condition is usually associated with diarrhoea and there is no tumour.

To sum up the diagnosis of pyloric stenosis rests on a few very definite symptoms and signs. If these are present

the diagnosis should be made confidently and treatment instituted without delay. Lack of decision on the part of the physician will endanger the child's life. A radiograph is unnecessary if the tumour has been felt, and filling the baby's stomach with barium is to be deprecated unless there are definite grounds for doubt in the diagnosis.

**Choice of Treatment** There are two main lines of treatment.

(1) *Medical* This is directed so as to lessen the spasm of the pylorus by medical measures while maintaining the child's strength till the spasm passes off as it tends to do after the end of the fourth month.

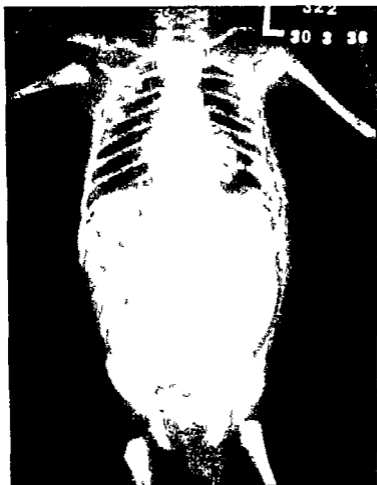
(2) *Surgical* By dividing the muscular coat of the pylorus to relieve the condition by surgical means.

In Scandinavia the medical line is favoured and very satisfactory results are claimed for it. Cases treated in this way will however require careful treatment and skilful nursing for from six to sixteen weeks during which time they must be safeguarded from all risk of intercurrent infection as their hold on life is very precarious. In the institutions at our disposal at present such a routine is very hard to obtain, and it is difficult to persuade parents to stand by idly for weeks or even months while the progress of their baby appears to them almost negligible. Hence we find that the treatment of choice is a combination of medical and surgical methods. We do not rush the child into the operating theatre as soon as the condition is diagnosed but rather correct his dehydration and generally improve his condition. When this is satisfactory the operation is performed after which he is nursed, not in the surgical but in the medical ward.

This line of action has proved very satisfactory and we have only lost one out of sixteen cases of pyloric stenosis so treated in the National Children's Hospital in the last couple of years.

**Medical Treatment** As we have already mentioned pyloric stenosis tends to undergo spontaneous cure after the fourth month of life. The hypertrophy does not then disappear but the lumen of the pylorus gradually increases in size and more and more food is able to pass through. Medical treatment aims at (1) controlling the spasm (2) reducing the swelling of the mucous membrane, (3) maintaining the child's strength (4) preventing intercurrent infection during what we may call the active period of the disease.

PLATE III



CASE 2B—PYLORIC STENOSIS

Two hours after meal No emptying Muscular effort (case 1

# PLATE IV



## CASE 3--FALLOU SPASM

Relatively small amount of barium had passed pylorus in one hour. Active peristalsis in stomach. Spasmodic contraction at jejunal segment.

Vomiting reduces the amount of body fluid and the chlorine content of the blood, producing dehydration and an alkalosis. Hence the first essential of treatment must be to restore the acid base balance and to supply fluid to the dehydrated tissues. This is simply accomplished by the administration of normal saline given intravenously, subcutaneously or intraperitoneally. If operation is to be performed in the near future the intraperitoneal route should not be used and the anterior abdominal wall avoided as a site for injection. Injections of normal saline, which also contains 5 per cent glucose for nutritive purposes will have to be given frequently if the baby's strength is to be maintained. Not infrequently the condition is only diagnosed after the baby has become very emaciated and dehydrated and has reached an almost moribund condition. Under these circumstances a serum transfusion is an invaluable therapeutic measure. The child being dehydrated, his blood is already over-concentrated, hence a whole blood transfusion, whereby more red blood corpuscles would be added to the baby's already over loaded circulation, is clearly contra indicated. The administration of adult serum is, however, an excellent method of helping the infant past this critical stage, and has been used with marked success by us on a number of occasions.

It has been shown that the longer the intervals between feeds the less the likelihood of vomiting, that small concentrated feeds are more likely to be retained than large dilute ones and that when the infant vomits if he is fed again immediately, he often retains the second feed. Hence Feed four hourly and give thickened feeds. If the child vomits re-feed at once. Breast milk with sugar added is the best feed for these babies, and every effort should be made to obtain it both for private and hospital cases. Where this is impossible an acid milk formula is usually satisfactory. Lactic acid or hydrochloric acid milk (either made up directly, or one of the proprietary acid milks with 10 per cent additional glucose) may be used satisfactorily, or a plain milk mixture containing 10-20 per cent starch.

Gastric lavage twice a day is a measure of real value for the condition. It helps the gastritis and removes the mucus from the dilated stomach. It appears also to lessen the irritability of the stomach and thereby the tendency to vomit. If much vomiting has taken place there will be a tendency to alkalosis,

hence it is unwise to wash out the stomach with a solution of bicarbonate as is often suggested. Normal saline should be used instead.

Atropine luminal eumydrin (atropinemethylnitrate) \* may be given in doses as described for the treatment of pylorospasm (p. 71). They reduce the nervous irritability, thereby lessening the spasm of the pyloric muscle and the tendency to vomit.

**Surgical Treatment** When the time for operation has been settled the following pre-operative routine should be carried out in the medical ward before the infant is sent down to the operating theatre —

(1) Give subcutaneously normal saline and glucose 5 per cent 1 oz 2 p.m. the day before the operation.

(2) Gastric lavage before 6 p.m. the same day.

(3) Subcutaneous saline and glucose 4 oz 2 a.m. day of operation.

(4) Gastric lavage half an hour before going to theatre, care being taken to empty the stomach.

**Operative Treatment** The only operation performed to day for the relief of congenital pyloric stenosis is that devised by Rammstedt and described by him in 1912. This procedure consists in exposing the hypertrophied pylorus incising the peritoneum over the tumour and then splitting the thickened tissues right down to the gastric mucosa. The technique employed in the National Children's Hospital Dublin follows closely that employed by Sir James Walton at the London Hospital.

The child is conveniently immobilised by being bandaged on to a well padded cross. Gas oxygen ether anaesthesia is used. A high right rectus incision is made—which stops short as a rule above the level of the umbilicus. By keeping the incision short and high its closure is made much easier as the greater part of it will be over the liver which has no tendency to bulge out and thus hamper closure. The peritoneum having been incised the dilated stomach is readily found and pulled gently to the left with the operator's left hand while his right index finger is used to hook up the pyloric tumour which is generally lying under cover of the liver. While the assistant

\* Since writing the above one of us (W.R.F.C.) has had the opportunity of using eumydrin in a number of cases of pyloric stenosis. The results have been most encouraging.

holds the body of the stomach the operator steadies the pyloric mass with the thumb and index finger of his left hand. The peritoneum over the whole length and in the long axis of the tumour is incised with a small sharp knife. The tumour itself is readily and safely split by making little stroking movements through its substance with the flat end of a blunt Durham's dissector. The splitting process is carried right down to the mucosa extreme care being taken not to perforate the latter particularly at the junction of the pylorus with the duodenum. The splitting process is conveniently completed by inserting a small artery forceps in the depths of the incision made with the dissector. When in position the blades of the forceps are gently opened. When the tumour has been adequately split the mucosa will bulge outwards towards the peritoneum. If it has been accidentally perforated a repair should be effected using fine catgut and a small needle. Omentum is not usually available to complete sealing off but should be employed if present. Haemorrhage is not likely to occur if the tumour is excised accurately in its mid axis but if it proves troublesome underpinning the bleeding point with a fine catgut stitch will prove probably the best course. The closure of the abdominal wall will present no difficulty if the incision has been made as described above. The whole operation takes about ten to fifteen minutes. The after care of the patient is a matter of extreme importance and is dealt with below.

**Post-operative Treatment** When operation for the relief of pyloric stenosis first came into use most cases came to the theatre in a state of great emaciation and starvation and elaborate methods of post-operative feeding were introduced to supply the child with the food and fluid which he so urgently needed. The routine shown on p. 78 which was used by Dr. Robert Hutchison in his ward at the Hospital for Sick Children, Great Ormond Street is a fairly usual example of this. It is our practice to follow this routine still though there is less reason for it now than formerly since with the improved diagnosis of to day the infants as a rule are in much better condition when they come to operation. Indeed on several occasions when a small nick was made accidentally in the duodenal mucous membrane at operation the whole post-operative routine was changed nothing being given by mouth for forty-eight hours while fluid was continued parenterally. The babies thus treated made complete recoveries.

## Post-operative Feed Schedule (after Hutchison)

1st	11 30 a m	$\bar{5}_1$	Glucose water 5 per cent	or	Glucose water 5 per cent	or	Glucose water 5 per cent
	12 noon	$\bar{5}_1$					
	12 30 p m	$\bar{5}_1$					
	1 0 p m	$\bar{5}_1$					
	1 30 p m	$\bar{5}_1$					
	2 0 p m	$\bar{5}_1$	$\frac{1}{2}$ strength breast milk		$\frac{1}{2}$ strength $\frac{1}{2}$ cream Cow and Gate		Skimmed Cow and Gate
	3 0 p m	$\bar{5}_1$					
	4 0 p m	$\bar{5}_1$					
	5 0 p m	$\bar{5}_1$					
	6 30 p m	$\bar{5}_1$					
	8 0 p m	$\bar{5}_1$	$\frac{1}{2}$ strength breast milk		$\frac{1}{2}$ strength $\frac{1}{2}$ cream Cow and Gate		Skimmed Cow and Gate
	9 30 p m	$\bar{5}_1$					
	11 0 p m	$\bar{5}_1$					
2nd	1 a m	$\bar{5}_1$	$\frac{1}{2}$ strength breast milk.				
	3 0 a m	$\bar{5}_1$					
	5 0 a m	$\bar{5}_1$					
	7 0 a m	$\bar{5}_1$					
	9 0 a m	$\bar{5}_1$					
	11 0 a m	$\bar{5}_1$					
	1 0 p m	$\bar{5}_1$					
	3 0 p m	$\bar{5}_1$					
	5 0 p m	$\bar{5}_1$					
	7 0 p m	$\bar{5}_1$					
	9 0 p m	$\bar{5}_1$					
	11 30 p m	$\bar{5}_1$					
3rd	2 0 a m	$\bar{5}_1$ $\bar{5}_1$	then $\bar{5}_1$ three hourly at the breast				
	4 30 a m	$\bar{5}_1$ $\bar{5}_1$					
	7 0 a m	$\bar{5}_1$ $\bar{5}_1$					
	9 30 a m	$\bar{5}_1$ $\bar{5}_1$					
	12 0 noon	$\bar{5}_1$ $\bar{5}_1$					
					then $\bar{5}_1$ three hourly $\frac{1}{2}$ cream Cow and Gate		

Both before and after operation the baby must be kept in an isolation ward so as to avoid intercurrent infection. Care should be taken not to feed the baby in the recumbent position (see p. 442) so as to lessen the risk of otitis media. Most infants will commence to put on weight immediately after operation but occasionally they appear to have lost the power of assimilation and fail to gain weight for a considerable period. This is particularly marked in cases in which the condition commences late or who have not been diagnosed early. In these cases the physician and nursing staff may be driven almost to despair before the baby commences to pick up. If an intercurrent infection occurs in these cases the outlook is very grave. However, hope should never be abandoned because occasionally after weeks or even months of failure a child will suddenly respond to treatment and commence to thrive. Recently in the National Children's Hospital such a case remained stationary or tended to lose weight for some two and a half months after

operation, being kept alive by many parenteral administrations of normal saline and glucose and three transfusions. Suddenly he commenced to gain weight, and in a few weeks made astonishing progress which he maintained, and was discharged eventually completely cured.

**Summary of Treatment Recommended** (1) As soon as the diagnosis is made relieve the dehydration by repeated administrations of normal saline and glucose 5 per cent given parenterally

(2) Commence gastric lavage twice a day with normal saline

(3) Feed with breast milk reinforced with dextrin maltose if possible, otherwise with acid milk and sugar or thickened feeds

(4) Re feed if vomiting occurs

(5) Give atropine luminal eumydrin by doses described on p 71

(6) As soon as the baby is in sufficiently good condition, if surgical treatment is decided upon, prepare him for operation as described on p 76

(7) Perform Rammstedt operation

(8) Follow feeding schedule, p. 78

(9) Keep isolated till discharged from hospital

## SECTION III

### CHAPTER IX

W R F COLLIS

#### BREAST FEEDING

(Advantages to Mother to Baby—Contra indications—Excretion of Drugs in Breast Milk Preparation of Mother for Lactation—Complementary and Supplementary Feeds—Method of Breast Feeding—Weaning—Difficulties Nasal Catarrh Cleft Lip and Palate Weakness Infections of Mouth, Pendulous Breast Breast Infections—Over feeding—Underfeeding—Quality of Milk )

BREAST feeding should be recommended to the mothers of all ranks of society as the best method of infant feeding Its advantages may be summarised as follows —

**A To the Mother** Breast feeding is a natural part of child bearing and is good for the mother's physical and mental state We know definitely that the stimulation of the breast by sucking reflexly encourages involution of the uterus That it is beneficial to her general health is also an undeniable fact, though this is more difficult to prove scientifically Certainly from the psychological point of view it is most desirable It helps the mother towards the realisation of motherhood and all it entails and is particularly important after her first pregnancy Up to the birth of her first baby she is often only a girl now suddenly she becomes a woman and the suckling of her infant child helps to bring about this profound psychological change in a unique way Apart from these deeper underlying truths there are certain simple advantages in breast feeding it is cheaper than artificial feeding, and simpler—no milk has to be bought or bottles prepared or washed

**B To the Infant** First of all breast milk is the best possible food for the infant The percentages (see next chapter) of carbohydrate fat and protein are those which are most easily digested and are provided in the breast milk in the most assimilable form Secondly the breast milk is naturally sterile Thirdly, it contains immune bodies which will help to protect the child before it is able to acquire its own active

immunity against disease. Fourthly it contains the vitamins which control satisfactory growth development and health of the growing tissues (see special chapter)

Whether or not suckling also has a good psychological effect on the infant is still somewhat a matter of dispute but it is undeniable that when a healthy mother feeds her own baby both benefit by health and happiness

Having said this it is necessary to issue a word of warning against becoming fanatical on the subject of breast feeding. I have seen nurses take up an almost moral attitude upon the matter and subject unfortunate mothers who cannot feed their babies for perfectly legitimate reasons to long tirades thereby upsetting the home and making any possibility of even partial breast feeding impossible. Our attitude must be scientific human and objective. We know that breast feeding is the right and best course but we must accept life as we find it. If we are dealing with a poor mother who is leaving the maternity hospital on the eighth day to return to her home where she is awaited by her husband and five other children who expect her immediately to resume control of the household which means cooking washing and managing generally for the lot of them if she is anæmic and under nourished and all but tired out we must not rant at her if she finds herself unable to feed the latest arrival. We must take a little trouble and try and get her extra nourishment through the social services and temporise by complementary feeding in the hope that her milk supply will increase later. Where this hope is belied we must boldly wean the baby and explain carefully how artificial feeding may be made satisfactory. With mothers in the higher ranks of society it is also necessary to be understanding and to be able to distinguish between the mother who genuinely cannot feed her baby due to lack of development of the mammary tissue and those who wish to evade the tediousness and lack of liberty that suckling a baby entails.

### Contra indications to Breast Feeding

If a mother has active tuberculosis it is most important that the baby be removed from all contact with her as soon as possible after birth for unless this is done the child will almost certainly contract the disease. Though a syphilitic mother may nurse her baby (Colles' law) and babies during the neo natal

period are immune to scarlet fever and diphtheria, the author feels that to insist upon breast feeding in such circumstances is to carry the doctrine too far, and in any case of severe disease an infant should be weaned and removed from contact with the mother during the active phase of the illness

Nephritis is a definite contra indication to breast feeding, as indeed are any of the severer toxæmias of pregnancy

Menstruation recommencing during lactation must not necessarily be regarded as an indication for weaning as although the reappearance of the first period is not uncommonly accompanied by an upset in the infant, this often clears up and when the new cycle has been established both mother and baby may continue to thrive

Pregnancy occurring during lactation is usually an indication to wean as the suckling of the infant may cause abortion

**Excretion of Drugs in Breast Milk** In the past it has often been taught that certain aperients such as cascara and senna were excreted in the mother's milk and hence that it was unwise to correct constipation in the mother by the administration of the vegetable group of aperients. Recent investigations have not borne out this theory however and it would now seem that any drug necessary for the mother's health may be prescribed without fear of upsetting the infant. Very small quantities of salicylates arsenic mercury and iodides may be excreted in the milk but not in sufficient quantities either to benefit or upset the infant. Belladonna decreases the secretion of all glands and should always be prescribed with caution to a nursing mother

**Preparation of the Mother for Lactation** *The Nipples* Preparation of the nipples should commence during the third month of pregnancy. They should be washed daily in cold water and spirit, and scrubbed with a soft brush so as to make them erect. If they are retracted they must be squeezed out manually each day or sucked out by a breast pump. Unless these measures are adopted early enough it may be impossible for the infant to suckle the breast when the time comes

**The General Health of the Mother** A satisfactory flow of good breast milk can be obtained only if the mother has been healthy during her pregnancy and has had an adequate diet during this period

Breast feeding should be begun as described on p. 80, and continued without addition until the fifth or sixth month. At

this stage additional feeding is recommended. Where possible some breast feeding should be continued until the ninth or even the twelfth month. Throughout the whole period breast feeding must be controlled by periodic test feeds (as described on p. 24). The indications for test feeding during these months are (1) Failure to gain weight (2) crying after feeds and sucking of fingers (3) green motions or (4) vomiting and wind. Green motions occurring in a breast fed baby are usually due to a hunger diarrhoea and point to the necessity of complementary feeds.

**Complementary and Supplementary Feeds** Complementary feeds are additional bottle feeds given at the end of the breast feed. Supplementary feeds are bottle feeds which replace one or more breast feeds in the twenty four hours. The former are much more satisfactory than the latter which usually lead to the drying up of the remaining breast milk in a few weeks.

### Method of Breast Feeding

Instruct the mother to cleanse the nipples carefully before every feed washing them with warm sterile water. Only one breast should be used at each feed. The mother turns to that side and gives the nipple in such a way that the child's nose is not forced against the breast and respiration obstructed. Sometimes the child falls asleep at the breast tell the mother to waken and stimulate him to suck. After the feed the child should be held up till the wind breaks as a certain amount of air is always swallowed which if not regurgitated may later lead to vomiting.

After feeding the nipple should be washed with borie lotion and spirit. If the nipples become cracked a nipple shield should be worn in the intervals between feeds. If the child cannot take the nipple this is due either to his inability to suck or because the nipple is unduly depressed. In either circumstances breast feeding should be persisted with for some time even if complementary feeds have to be given as the child's sucking powers will often improve.

**Weaning** As already stated complete weaning is not necessary till the twelfth month. Many mothers find such prolonged breast feeding impossible and the child has to be weaned at an earlier date. At any time the process should be deliberate.

The following is the routine which may be adopted —

When the child reaches six months or 10-16 lb in weight, is about the best time to start making additions

(Warning Avoid weaning during the hot weather, especially from July to September It is better to breast feed the baby for longer than the usual time than to wean during these months)

The following additions may be made at intervals of one week —

1st Stage Give 2 tablespoonfuls whole milk (sweetened) before the 2 p m feed

2nd Stage Thicken with potato or cereal

3rd Stage Give a feed of groats (one third to one-half a teacupful) made with milk at the 10 a m feed

4th Stage Give one third to one half a teacupful of some cereal preparation such as patent barley cream of rice Sister Laura's Food Irish Cornflakes Nue va Farex etc made with milk at the 6 p m feed

5th Stage Add one or two heaped eggspoonfuls of raw egg yolk at the 10 a m feed

About the ninth or tenth month completely replace breast feeding by the artificial method substituting whole cow's milk (boiled) for breast milk and putting the child on Diet Sheet No 3 (see p 95)

**Treatment of the Breasts at Weaning** If weaning is carried out as above the breasts usually give very little trouble If uncomfortable swelling occurs draw off some of the milk by expression or pump and firmly strap the breasts

### Special Points

(1) *Boiling of Milk* As soon as it comes boil the milk for three minutes cool it by standing milk jug in water cover and keep in a cool place until ready for use

(2) *Giving of Orange Juice* Give a tablespoonful of orange or tomato juice daily with the addition of water and sugar

(3) *Giving of Cod liver Oil* If possible give a teaspoonful of cod liver oil twice a day as soon as the child is weaned

## COMMON DIFFICULTIES IN BREAST FEEDING

(1) **Suckling** The baby may find suckling difficult due to —

(a) *Nasal Catarrh* Infants not uncommonly receive nasal infection at birth or shortly afterwards and the mucous membrane becomes engorged mucus is secreted and some nasal obstruction occurs Hypertrophy of adenoid tissue is also not uncommonly found at birth and may lead to partial nasal obstruction The treatment is symptomatic

(b) *Cleft Lip and Cleft Palate* (see p 363) In severe cases of these conditions suckling may be impossible and the baby may have to be fed by spoon or gavage

(c) *Weakness* In premature and weakly babies suckling may be impossible In these cases where possible the breast milk should be drawn off by breast pump and given to the child by other means (see section on prematurity) Often these babies gain strength rapidly and will be able to take the breast satisfactorily after a few days Hence every effort should be made to maintain the mother's milk secretion

(d) *Infections of the Mouth* These and their treatment have already been mentioned (see p 63) Here again every effort should be made to maintain the mother's milk secretion while the mouth is being cleaned up The baby's mouth is often so painful in these cases that the breast is refused Hence in any case where a history of sudden refusal to feed is met with the baby's mouth should be examined immediately

(e) *Pendulous Breast* In mothers with large and pendulous breasts the baby may be almost suffocated while attempting to suck In these cases the mother must be instructed to support the breast when offering it to the baby and to see that the baby's nose is not obstructed while feeding

(2) **Breast Infections** (a) *Fissures of the nipple* may prevent suckling particularly if they become infected and painful If the preparatory measures given earlier in this chapter are followed cracked nipple should not occur If excoriation of the nipple takes place treat at once with some mild ointment (such as oil ricini tinc benzoin co equal parts) so as to prevent cracking When fissures occur keep nipple dry and bath freely with 20 per cent boric or other disinfectant lotion In severe cases cauterise with silver nitrate Pain may be very severe Hence when it is important to maintain the breast secretion use a nipple shield and cocaine the nipple before feeds for a few days while every effort is made to clear up the condition

(b) *Acute Mastitis and Breast Abscess* These conditions usually follow infection through a cracked nipple The breast becomes hot and painful and there is a general reaction with associated fever depending in degree upon the severity of the infection It is wise in these cases to take the baby off the affected breast at once The breast pump should not be used but the congestion relieved by hot fomentations and bella donna plasters Calomel gr 1 followed by a large dose of salts

may help to relieve the congestion in these cases. If pus forms surgical incision becomes necessary. The important consideration from the baby's point of view is that the condition often clears up satisfactorily in a short time and he may be put back on the breast with safety, normal secretion being restored once more.

### Quantity of Feed

*Overfeeding* in breast fed babies is common. The infant is usually brought to the doctor for vomiting and too numerous stools. On examination an overweight fat baby is found. If the motion is examined it will be found to be of normal colour. The diagnosis rests upon the test feed, which should always be performed immediately in such cases.

*Treatment* If the baby is being fed three hourly the interval should be lengthened to four hours a little water should be given before each feed and only one breast used at a time. The actual amount which the baby should get must be calculated and test feeds performed till the correct time which the baby takes to acquire this amount discovered, and thereafter the infant only fed for this specific time.

### Underfeeding

This is the commonest difficulty encountered in breast feeding. It is characterised by failure to thrive, colic, associated first with constipation and then frequent green motions. Most commonly the baby is brought to the doctor for diarrhoea the mother having become alarmed at the green motions. True diarrhoea is very seldom seen in breast fed babies, and when it does occur is usually due to parenteral infection. Hence in every breast fed baby suffering from "green diarrhoea" a test feed should be done immediately. If, as is usually the case, the baby is found to be getting an insufficient supply of breast milk complementary feeding and the measures for increasing the supply of breast milk as described on p. 24 should be started without delay. This procedure often clears up the condition in a few days.

### Quality

Certain babies occasionally appear unable to digest their mother's milk. Test feeds show that they are getting sufficient

quantity per feed, analysis of the milk reveals no abnormality of quality, yet the baby gets colic, vomits or has diarrhoea, screams and fails to gain weight. It has been said that this condition tends to occur in certain over anxious and nervous mothers. A continually screaming baby, however, always produces over anxiousness in a very short time, so it is difficult to dogmatise in these cases as to which is the primary cause. Into this group falls one definitely recognisable type. Breast milk with its relatively high fat and carbohydrate content produces an acid stool, while cow's milk produces an alkaline stool on account of its higher protein value. Certain infants appear to be hypersensitive to an acid stool and get diarrhoea and show excoriation of the buttocks on pure breast feeding, but if the stools are made alkaline by the addition of complementary feeds of cow's milk or casein they clear up at once.

Hence in this general group of unsatisfactory breast fed babies it is always wise to try the effect of giving a couple of complementary feeds per day before abandoning breast feeding. If the test feed shows that the baby is getting enough, the length of time the baby is allowed at the breast should be shortened, and the appropriate amount of cow's milk given after the breast milk instead. If this measure fails it is best to wean the baby on to a satisfactory milk formula without further delay, unless some other cause such as a parenteral infection, can be shown to be the source of the trouble.

# CHAPTER X

## W R F COLLIS

### ARTIFICIAL FEEDING

#### The Normal Baby

(Breast Milk—Cow's Milk—Protein—Fat—Carbohydrate—Vitamins—Buffer Substances—Immune Bodies—Salts—Choice of Infant Food—Modified Cow's Milk Method Diet Sheets 0-6 Months 9-12 Months 1 2 Years 2-5 Years—Caloric Feeding)

A THOROUGH knowledge of the composition of breast milk and the importance of each factor in it is essential before any attempt can be made to approach the subject of artificial feeding

Below is given a table in which the constituents of breast milk are compared with those of boiled cow's milk in detail

Breast Milk			Boiled Cow's Milk
Protein	Casein	1 per cent approx	2.5 per cent approx
	Lactalbumen	1	1
Fat		3.5	3.5
Carbohydrate		7	4
Vitamins		+	—
Buffer substances		+ —	+
Immune bodies		+	—
Salts		0.00 per cent	0.75 per cent
Iron		0.001 gm per 1000 c c	0.000 gm per 1000 c c

Let us first examine this table in detail and then proceed to discuss the various methods of artificial feeding in general use in the light of knowledge thus gained

(1) The Protein Cow's milk has 1.5 per cent (approximately) more protein than human milk the additional protein being in the form of casein. Of the two main proteins of milk lactalbumin is by far the most soluble and does not form a curd on the addition of an acid. Casein on the other hand is most insoluble and forms a large thick curd in the stomach when mixed with the gastric hydrochloric acid. It is for this

reason that plain unboiled undiluted milk is not suitable for many infants

(2) **Fat** The percentage of fat in breast milk and shorthorn cow's milk is nearly identical but the size of the fat globule is considerably larger in cow's milk. Kerry and Jersey cows yield a milk very much richer in fat than shorthorn cows. It may sometimes even be as high as 8 per cent and some modification is essential before such milk can be fed to infants.

(3) **Carbohydrate** Breast milk contains approximately 2.5 per cent more lactose than cow's milk. Carbohydrate in the form of the simple sugars is the most easily digested of all foods. It is absorbed from the stomach and upper part of the small intestines with great rapidity and is used largely in the production of energy. The more energetic the infant is therefore the more sugar he is likely to require. In any case he will require more than the normal amount present in cow's milk.

(4) **Vitamins** As we shall show later milk is always best boiled except when it is a Certified Tuberculin Tested Milk. As the supply of the latter is limited and more expensive we may assume that the ordinary consumer will use uncertified milk which has been boiled. Boiling destroys vitamins and hence boiled cow's milk is deficient in the necessary factors. No misgiving need be felt on this score however once the principle is grasped as substitutes can easily be added to the child's diet e.g. vitamins A and D in some form of cod liver oil or synthetic substitute vitamins B and C in orange or other fruit juice (see later chapter).

(5) **Buffer Substances** These are chiefly phosphates and proteins. They exist in cow's milk in a higher percentage than in human milk. They tend to neutralise the natural acidity (HCl) of the child's gastric juice thus rendering digestion more difficult.

(6) **Immune Bodies** The mother passes on a good deal of passive immunity to her infant through the colostrum and breast milk. These substances which may be of great importance in helping the child to resist infection during its first few months of life are absent in cow's milk and this fact constitutes one of the main arguments for persevering with breast feeding even under difficult circumstances.

(7) **Iron** Human milk to some extent and cow's milk to a greater extent, are deficient in iron and infants fed for more

than six months without any addition to the diet tend to become anæmic as the iron which has been stored in their livers during foetal life becomes used up by this time

### Choice of Infant Food

Many different ways of infant feeding are advanced in different parts of the world the advocates of which all proclaim the success of their particular method. Some announce their system almost with religious fervour and instil into their disciples the belief that by their system only may children be fed properly. In England the vendors of patent infant foods and dried milks have made great headway by elaborate advertising campaigns and clever travellers till in some parts of the country no mother would think of feeding her infant on ordinary modified cow's milk but will feel it her duty to buy some expensive tinned patent food. Worse still the problem of infant feeding has come in many places to be regarded as a sort of game—find the suitable mixture! So that it is not uncommon for a child to be brought to a consultant physician after the feeding has already been changed five or six times from one proprietary mixture to another when in reality the baby is suffering from some definite complaint such as pyloric stenosis. This attitude is most deplorable and leads to many of the disasters which occur during the first year of life. The truth is that the normal baby can be fed successfully on most of the systems advocated. The reverse is equally true. The 'difficult baby' cannot be fed on any system successfully till his particular idiosyncrasy has been diagnosed and dealt with. Let us give an example—if a baby has a fat idiosyncrasy and cannot tolerate the usual amount of fat changing it from one whole milk to another or feeding four hourly instead of three will do no good. But once the cause is diagnosed and a low fat feed instituted the baby will begin to thrive.

The subject of infant feeding is one about which many books have been written and hence it is only possible here to outline the method of feeding which the author has found suitable in Dublin and to indicate briefly how it can be modified to meet the special needs of certain cases.

**The Modified Cow's Milk Method.** Fresh cow's milk is many times cheaper than any dried or proprietary milk its composition is known to all and when later additions have to be made to the diet it is a most suitable base for thickened feeds.

giving extra fluid as the child does not thrive unless it gets at least 2½ oz of fluid per pound per day. For this reason alone it would be wise to add water to milk before feeding. We have already seen however that it is wise to dilute the milk so as to give a 2/1 milk water mixture. If this is done we will be giving approximately the right amount of milk and also fluid to the child.

*Example* A child of 10 lb requires  $10 \times 1\frac{1}{2} = 15$  oz of sweetened milk per day plus 10 oz of water.

To simplify the matter still further our ordinary formula is calculated on the basis that the infant requires 2½ oz of a 2/1 milk water mixture plus one level teaspoonful of sugar per pound of body weight per day. This is quite sufficiently accurate for the average infant and the following diet sheet is used to assist mothers during the first six months of life.

### DIET SHEET No 1

(To Feed a Normal Baby from Birth to Six Months Old)

Feed baby at 7 10 1 4 7 and 10 p m  
7 11 3 7 and 11 p m

At each feed give baby      oz or      tablespoonfuls of this mixture —

Boiled cow's milk 1 pint  
Water                      ½ pint  
Sugar 3 level tablespoonfuls

### Special Points

(1) The milk should be boiled for three minutes and should not be too creamy.

The water which is added should also be boiled.

(2) See that the hole in the teat is a good size so that the baby can get the feed in twenty minutes or less.

(3) Hold baby up after each feed until the wind breaks.

(4) The milk mixture should be kept covered up in a cool place during the day in a jug which has been scalded before use.

(5) After the end of the third week give orange or tomato juice two or three teaspoonfuls diluted with a little water and sweetened with sugar daily.

(6) Unless not tolerated give a small teaspoonful of cod liver oil before one increasing to two of the feeds.

(7) Increase the size of each feed by ½ oz or one tablespoonful each fortnight until baby is taking a 7-oz feed or 14 tablespoonfuls. After that any further increase should be made following the instructions given on Diet Sheet No 2.

be, the mother should hold up the child after each feed while stroking him gently on the back from time to time till the air is regurgitated once or twice

(4) It is most important that the milk shall not become re-infected after being sterilised. Hence flies and dust must be kept from falling into the milk jug. The latter should be scalded before use each morning and carefully cleansed each night

(5) We are aware that scurvy is not seen clinically till the fifth month, but recent work suggests that sub clinical scurvy may occur much earlier, hence recently we have introduced orange juice after the third week. It is usually well tolerated and appears beneficial

(6) Cod liver oil is introduced as early as possible to supply vitamins A and D

(8) Egg yolk we have found of great value if there is a tendency to anemia. It is rich in vitamins, contains iron, and is usually well tolerated though it is always well to introduce it gradually as some infants tend to get loose motions or vomit if it is given in too large amounts at first

### DIET SHEET No 2

(For Infants from Six Months to Nine Months Old or  
Weight 15 to 18 lb )

7 a m	6 oz milk (boiled) 1 level teaspoonful sugar
10 a m	6 oz milk (boiled) 1 level teaspoonful sugar Add 1 or 2 teaspoonfuls raw egg yolk
2 p m	6 oz milk (boiled) 1 level teaspoonful sugar One, increasing to two heaped teaspoonfuls of potato, to which a small quantity of butter has been added, or groats or other cereal
6 p m	Same as 7 a m with the addition of rusk or finger of toast towards the end of this period
10 p m	Same as 7 a m

### Special Points

(1) *Boiling of Milk* The milk should be brought to the boil (till bubbles appear) then cooled by standing it in cold water, covered up and kept in a cool place until ready for use

(2) *Giving of Orange Juice* The juice of half an orange with water added and sweetened with sugar should be given daily. This may conveniently be given between 8 and 10 a m or before 7 p m feed

(3) *Giving of Cod liver Oil* If possible, give a teaspoonful of cod liver oil before three of the feeds

- (4) As whole milk is being given it is particularly important to give two bottles of boiled water to the baby every twenty four hours
- (5) Towards the end of this period cereal should be added at the 10 a m feed

When a baby reaches 15 lb it usually needs additions to the diet. By now also it usually tolerates whole milk. The above diet sheet has been recently introduced, the previous sheet being considerably modified. Due to recent work, the introduction of cereals in any quantity before the ninth month is not to be recommended as a general principle. At the same time, other work suggests that more than 25-28 oz of milk per day has an unfavourable influence on the absorption of calcium and iron. Also the bone and vegetable soup so strongly advocated by Pritchard and Paterson has been found on analysis to contain few nutritive constituents and to be almost valueless as a food. Hence we attempt to substitute with raw egg yolk and potato as far as possible. The latter is a highly nutritive substance rich in proteins and vitamins as well as carbohydrates and if "floured" makes an excellent food for babies at this age. In the case of babies who are not satisfied or who fail to gain weight, additional cereals will have to be added to this diet.

In criticism of this diet it may be said that it is perhaps too rich in fat and that all babies will not tolerate whole milk even at this age. As a general routine however, it appears adequate though in certain cases the doctor will have to modify the milk mixture and perhaps reduce the egg or cut out the butter and make up with increased carbohydrate.

In regard to the *Special Points* little need be said—not more than 1 oz of orange juice should be given (the amount of juice obtainable from different types of oranges is very variable).

### DIET SHEET No 3

(For Infants from Nine to Twelve Months or Weight 18 to 21 lb approx.)

7 30 a m	BREAKFAST 6 oz milk (boiled) Half a cupful of porridge or groats Jacob rusk or toast and butter four mornings, half one egg other mornings, toast fried in bacon fat
11 a m	6 oz milk (boiled)
1 30 p m	DINNER 6 oz milk (boiled) One to three teaspoonfuls of pounded fish, scraped beef, mashed chicken rabbit or

be the mother should hold up the child after each feed while stroking him gently on the back from time to time till the air is regurgitated once or twice

(4) It is most important that the milk shall not become re infected after being sterilised. Hence flies and dust must be kept from falling into the milk jug. The latter should be scalded before use each morning and carefully cleansed each night

(5) We are aware that scurvy is not seen clinically till the fifth month but recent work suggests that sub clinical scurvy may occur much earlier hence recently we have introduced orange juice after the third week. It is usually well tolerated and appears beneficial

(6) Cod liver oil is introduced as early as possible to supply vitamins A and D

(8) Egg yolk we have found of great value if there is a tendency to anæmia. It is rich in vitamins contains iron and is usually well tolerated though it is always well to introduce it gradually as some infants tend to get loose motions or vomit if it is given in too large amounts at first

### DIET SHEET No 2

(For Infants from Six Months to Nine Months Old or  
Weight 15 to 18 lb )

7 a m	6 oz milk (boiled) 1 level teaspoonful sugar
10 a m	6 oz milk (boiled) 1 level teaspoonful sugar Add 1 or 2 teaspoonfuls raw egg yolk
2 p m	6 oz milk (boiled) 1 level teaspoonful sugar One increasing to two heaped teaspoonfuls of potato to which a small quantity of butter has been added or groats or other cereal
6 p m	Same as 7 a m with the addition of rusk or finger of toast towards the end of this period
10 p m	Same as 7 a m

### Special Points

(1) *Boiling of Milk* The milk should be brought to the boil (till bubbles appear) then cooled by standing it in cold water covered up and kept in a cool place until ready for use

(2) *Giving of Orange Juice* The juice of half an orange with water added and sweetened with sugar should be given daily. This may conveniently be given between 8 and 10 a m or before 7 p m feed

(3) *Giving of Cod liver Oil* If possible give a teaspoonful of cod liver oil before three of the feeds

(4) As whole milk is being given it is particularly important to give two bottles of boiled water to the baby every twenty four hours

(5) Towards the end of this period cereal should be added at the 10 a m feed

When a baby reaches 15 lb it usually needs additions to the diet. By now also it usually tolerates whole milk. The above diet sheet has been recently introduced the previous sheet being considerably modified. Due to recent work the introduction of cereals in any quantity before the ninth month is not to be recommended as a general principle. At the same time, other work suggests that more than 25-28 oz of milk per day has an unfavourable influence on the absorption of calcium and iron. Also the bone and vegetable soup so strongly advocated by Pritchard and Paterson has been found on analysis to contain few nutritive constituents and to be almost valueless as a food. Hence we attempt to substitute with raw egg yolk and potato as far as possible. The latter is a highly nutritive substance rich in proteins and vitamins as well as carbohydrates and if floured makes an excellent food for babies at this age. In the case of babies who are not satisfied or who fail to gain weight, additional cereals will have to be added to this diet.

In criticism of this diet it may be said that it is perhaps too rich in fat and that all babies will not tolerate whole milk even at this age. As a general routine however it appears adequate, though in certain cases the doctor will have to modify the milk mixture and perhaps reduce the egg or cut out the butter and make up with increased carbohydrate.

In regard to the *Special Points* little need be said—not more than 1 oz of orange juice should be given (the amount of juice obtainable from different types of oranges is very variable)

### DIET SHEET No 3

(For Infants from Nine to Twelve Months or Weight 18 to 21 lb approx )

7 30 a m	BREAKFAST 6 oz milk (boiled) Half a cupful of porridge or groats Jacob rusk or toast and butter four mornings, half one egg other mornings toast fried in bacon fat
11 a m	6 oz milk (boiled)
1 30 p m	DINNER 6 oz milk (boiled) One to three teaspoonfuls of pounded fish, scraped beef, mashed chicken, rabbit or

	well boiled tripe or 1 to 2 tablespoonfuls of potato and a little butter, stewed fruit or junket or custard
5 p m	TEA 6 oz milk (boiled) rusk toast, bread and butter Towards the end of this period some cereal may be added to this meal or egg (on those days when egg is not given at breakfast) or potato (when not given for dinner)
10 p m	4 oz milk (boiled)

### Special Points

(1) Boiled water should be offered to the child between meals, particularly between dinner and tea This is of the greatest importance during the hot weather

(2) *Boiling of Milk* The milk should be brought to the boil (till bubbles appear) then cooled by standing in water covered up and kept in a cool place until ready for use

(3) *Giving of Orange Juice* A tablespoonful of orange or tomato juice should be given daily with water and sugar added to sweeten It is best given a short time before the baby's breakfast

(4) *Giving of Cod liver Oil* A teaspoonful of cod liver oil should be given twice a day

(5) Feed from eight months from a cup as much as possible

(6) The additions suggested here must be made gradually from the previous diet sheet

(7) *Cereal* This should be well cooked before use

When the baby reaches 18-21 lb the addition of cereal becomes necessary and the principle of three main meals in the day should be introduced gradually We have also found that meat foods if presented in a digestible form can be introduced with advantage during this period and are better for growth and development than a high cereal diet Again we recommend rather more milk than is sometimes advocated If the child shows a tendency to ketosis or if the stools become pale and slimy, the milk will have to be reduced and the carbohydrate increased but in our experience this is seldom necessary

It is especially important to give additional water at this period The other special points are as before

### DIET SHEET No 4

(For Children from One to Two Years Old)

7-7 30 a m	Orange juice or grape juice or grape fruit juice (one dessertspoonful diluted with water and sweetened with sugar)
7 30-8 a m	BREAKFAST Porridge or groats (one small cupful), or bread and butter, toast, Jacob's rusk in milk Lightly boiled egg with breadcrumbs or finger of

toast fried crisply in bacon fat, or small rasher of *crisp bacon, or pounded plaice or sole*

Rusks or crisp toast

Milk, 6-8 oz (boiled), including that given with the above

11 a m Milk, 6 oz (boiled)

12 30-1 p m DINNER Pounded fish (boiled or steamed), or pounded chicken, rabbit, tripe, brains, etc., or scraped raw or underdone steak, or Irish stew with meat very finely chopped up (1 level tablespoonful) Boiled, baked, or mashed potato (1 rounded tablespoonful with butter and gravy)

Sieved sprouts, cabbage, spinach, cauliflower, parsnips, turnips, etc (1 level tablespoonful)

Milk pudding, or junket, or custard (1 tablespoonful, with the addition of a little stewed apples or prunes) or over ripe banana with a little milk (1 tablespoonful)

Milk, 6 oz (boiled), over pudding or as junket, etc

*Water to drink*

1 30-5 p m TEA Rusks or crisp toast, or stale bread, or stale sponge cake (with a little butter and honey or seedless jam) or junket, or custard, or mashed over ripe banana with a little milk

Milk, 6-8 oz (boiled), including that used with above

### Special Points.

(1) Give 1 teaspoonful of cod liver oil before three of the meals

(2) It ought not to be necessary to give anything after tea. If the child requires a drink before being put to bed, a portion of the 6-8 oz milk allowed at tea should be utilised and may be diluted with water

(3) Water may be offered to the child between meals, especially between dinner and tea

(4) The transition from the previous diet to the above one should take place gradually, each fresh item being introduced step by step

(5) Raw fruit should not be given except orange juice or over ripe banana. Fruit should be stewed and preferably sieved

(6) Only plain sugar sweets (barley sugar) may be given

### DIET SHEET No 5

(Diet from Two to Five Years of Age)

8-8 30 a m BREAKFAST Orange juice, or grape juice or grape fruit juice (1 dessertspoonful) diluted with water and sweetened with sugar

Porridge or groats (1 or 2 heaped tablespoonfuls) or grape nuts (powdered or well chewed by the child) with milk.

Egg (three mornings) or small rasher of crisp bacon with a little toast fried crisply in bacon fat (three mornings) or fish (one morning)

Crisp toast or rusk or stale bread with butter

Milk 6-8 oz (boiled) to include with milk given above

12.30-1 p.m. **DINNER** Cutlet or mince or stew or underdone beef or steak (finely cut up) or brains or fish or pounded chicken or rabbit (1 tablespoonful of any of these which should be varied as far as possible)

Potato (1 tablespoonful)

Finely mashed cauliflower or carrots or spinach or brussels sprouts etc (1 tablespoonful)

Milk pudding or junket or custard (1 heaped tablespoonful) with a little stewed fruit or jelly or seedless jam or mashed over ripe banana

Milk 6 oz (boiled)

*Water to drink*

4.30-5 p.m. **TEA** Wholemeal or brown bread (crisply toasted or stale) with butter and a little honey or seedless jam sponge cake or other plain cake may be given occasionally

Pudding or junket or custard etc as at dinner

(This may be omitted if appetite for puddings is poor)

Milk 6-8 oz (boiled)

6.30 p.m. **SUPPER** Milk 4-6 oz (boiled) diluted with water

### Special Points

(1) If possible the 6.30 meal should be left out and nothing given after tea

(2) Water not milk may be offered to the child between meals especially between dinner and tea

(3) As the child gets older the helpings of food may be increased Any increase in eggs and milk must be made cautiously

(4) After the third year the milk may be flavoured with a little tea or cocoa

(5) Sweets such as milk chocolate or barley sugar may be given immediately after tea but never at irregular hours

(6) Raw fruit must be given with care and is not recommended before the third year (This does not apply to over ripe bananas which is a well-digested food when given in suitable quantities) *Fruit is generally best given stewed*

(7) Instruct the child to chew thoroughly a habit which will stand to it in later life

(8) It is most important that cod liver oil should be continued over this period 1 teaspoonful being given in the day before three of the feeds

These diet sheets take us somewhat outside the age period for this work but are included for the sake of completeness They

are generally recognised standard diets for these ages except that we recommend rather more milk than Paterson and others, who, we feel, underestimate the advantages of whole milk in growth and overestimate the number of children whose digestion rebels against this size milk diet. Again, in special cases the quantity of milk may have to be reduced, but as a general rule we believe that these diets are better than those in which less milk and more cereal is given.

In Ireland there is no doubt that children brought up on potato, butter and milk are healthier than those whose diet contains large quantities of cereals.

Much further work is required upon the dietetics of these early years of life before this controversy can be settled, we believe, however with Professor Mellanby, that more and more stress will be laid on the protective food substances (*e g*, milk, eggs, meat, potatoes green vegetables etc) at the expense of the merely energy yielding foods (*e g* milled cereals, sugar, etc) as time goes on.

### CALORIC FEEDING

A common method of calculating diet formulæ for infants is to express the food value in heat units or calories.

One calorie	= the amount of heat necessary to raise 1 gram of water 1 degree centigrade
One gram protein	= 4.1 calories
" " carbohydrate	= 4.1 "
" " fat	= 9.3 "

Therefore in the case of human milk the calculation is as follows —

Protein	2	$\times 4.1 =$	8.2
Fat	3.5	$\times 9.3 =$	31.5
Carbohydrate	7	$\times 4.1 =$	28.7
Total			<u>68.4</u>

Therefore 100 gm of milk contain 68.4 calories

As there are 3.5 gm in 1 oz therefore —

$$1 \text{ oz milk contains } \frac{68.4}{3.5} = 19.5 \text{ calories}$$

1 oz of sugar contains 120 calories

As we have stated above, it has been found that the normal baby requires approximately 45–50 calories per pound of body

weight per day. We may therefore calculate the caloric requirements of a normal 10 lb baby as follows —

The baby will require  $10 \times 40 = 400$  calories per day

Suppose we decide to feed him on cow's milk then it will be necessary to add not less than 1 oz. of sugar to the milk

1 oz. of sugar — 120 calories

Therefore he will require  $400 \text{ less } 120 = 330$  calories in the form of milk. If 20 calories approximately are contained in 1 oz. milk therefore 330 calories approximately are contained in 17 oz. milk.

Hence he will require 17 oz. of cow's milk to which 1 oz. of sugar has been added. When the necessary additional water has been added it will be found that the final result approximates closely to the general 2 milk : 1 water + sugar mixture used as our standard feeding formula.

The diets included here and elsewhere are those used in the paediatric department of the Rotunda Hospital. They were founded upon Dr. Donald Paterson's method, modified subsequently to meet local needs and recent developments in dietetics.

## CHAPTER XI

W R F COLLIS

### PROBLEMS OF INFANT FEEDING

(Dyspepsia Due to—Carbohydrate Problem of Starch Digestion—Protein Dilution of Milk Citration and Peptonisation Dried Milks—Fat—Parenteral Infection (Otitis Media Pyelitis etc.)—Enteral Infection—Gastro enteritis and Treatment Acidified Milk Mixtures Protein Milk Whey Dyspepsia Due to Anæmia )

THE difficulties which may be encountered in infant feeding can be divided into two main groups

#### A Simple Dyspepsias Due to Food Idiosyncrasy

(1) *Carbohydrate* (2) *Protein* (3) *Fat*

#### B Dyspepsias Due to Infections

(1) *Parenteral* (2) *Enteral*

### A SIMPLE DYSPEPSIAS

#### (1) *Carbohydrate*

As has already been stated sugar is the most easily digested constituent of milk and may even be tolerated in a percentage double that found normally in breast milk. Nor does the type of sugar make very much difference. A lot of unnecessary fuss has been made in the past over the different types of sugar. But in our experience infants tolerate cane sugar just as well as lactose. Perhaps dextrin maltose is somewhat more assimilable than any other form and in consequence if it becomes necessary (as in the feeding of certain premature infants) to increase the percentage of sugar above the normal this preparation may be advised. Very occasionally a baby is found to have an idiosyncrasy against carbohydrate and cannot tolerate even the normal percentage of sugar in the feed. Every time sugar is added the infant loses weight and gets loose green, acid motions. These cases are extremely rare and loose motions due to other causes must not be placed under this heading. The diagnosis is always a difficult matter and can

usually only be made by careful observation of the baby by a skilled dietitian. *The treatment* for pure carbohydrate dyspepsia consists in giving a high protein feed such as Mead's protein milk or by adding casein (a powder containing 88 per cent calcium caseinate) to the feed.

The usual form of carbohydrate dyspepsia however is caused by the addition not of sugar but of starch to infants' diets at a too early age. It has been recognised by the laity for many years that the addition of a starchy solution to a milk mixture tends to prevent vomiting in certain infants. This is due to the fact that the addition of such substances to hot or boiling milk breaks up the protein curd of the milk to a very considerable degree. Starch is not digested and absorbed in the stomach and upper part of the small intestine as are the sugars but further down the alimentary tract. A good deal of it may reach the large intestine unchanged and here set up fermentation. This is particularly likely to occur if crude starch preparations are used such as the well known Dublin poor mother's receipt of the top of the gruel.

The symptoms which arise in these cases of carbohydrate dyspepsia are Loose frothy acid motions green or green yellow in colour flatulence and diarrhoea with occasional vomiting as the condition becomes worse.

**Treatment** Get the baby's intestine well cleared out by a bowel washout and a dose of castor oil. Give nothing but water for twenty four hours and then commence feeding on a simple milk mixture either a dried milk such as Trufood, Glaxo or half cream Cow and Gate or a boiled partially skimmed cow's milk. If this method is followed the ordinary simple carbohydrate dyspepsia will clear up almost at once and the baby commence to gain weight again immediately. If after applying the above measures the condition does not improve at once the physician should always check up his diagnosis as under these circumstances some additional cause such as a chronic otitis media is usually found to be present as well.

## (2) Protein

Protein dyspepsia is due to the large casein curd of cow's milk. It is never met with in breast fed infants.

**Symptoms** There is usually a variable amount of vomiting the vomit being full of thick curds. The motions are alkaline.

undigested, grey or green yellow in colour and contain undigested casein curds. They are usually loose but sometimes constipated. Protein digestion is a very variable factor, and whereas many infants even premature will tolerate whole milk, many others will not. Hence so as to meet the requirements of the average infant, our general principle is to dilute our standard milk mixture so as to make a milk 2/1 water dilution. We also order the milk to be boiled partly so as to assure sterility and partly because boiled milk forms a smaller curd in the stomach than unboiled milk. However, even this modification is not always sufficient, and protein dyspepsia not uncommonly occurs among infants fed on this 2/1 formula.

**Treatment** When the diagnosis of protein dyspepsia has been made it is usually wise to wash out the baby's stomach and give a dose of castor oil. (We are aware that some physicians say this is unnecessary but we have certainly found this procedure of value in the Rotunda Clinic.) The diet must then be corrected so as either to give less protein to the infant or present the protein in a more assimilable form.

The following methods are used —

(1) *Give less protein by diluting the milk* half and half with water and adding additional sugar. This may be sufficient in the milder cases.

(2) *Citrating the milk*. A solution of sodium citrate gr. ii. to gr. iii. is added to the feed. It is supposed that this method produces smaller and softer curds in the milk mixture. Lime water is used in a similar way. In our experience however this method usually fails in cases of dyspepsia due to casein curds.

(3) *The addition of cereal*. A very small quantity of a dextrinised cereal such as a teaspoonful of Sister Laura's food, Savory and Moore's food, etc., added to a pint of boiled milk will reduce the curd formation very considerably and is a good method of dealing with the condition. Crude starchy mixtures (e.g., "the top o' the gruel") must be avoided however, particularly in young infants—see section on carbohydrate dyspepsia.

(4) *Peptonisation*. Add requisite amount of Bengers' peptonising powder (containing pancreatic extract) to milk mixture which has been warmed to 98° F. Allow to stand for twenty to thirty minutes and then bring to the boil, thus terminating the digestive process by destroying the pancreatic enzymes. This is one of the best ways of dealing with bad

cases of protein dyspepsia but it should not be employed for more than a month's duration and the necessity for the addition of vitamins to the diet must not be forgotten while this treatment is being employed

(5) *Employ a dried milk* such as Glaxo Trufood, Cow and Gate Dun's milk etc. In mild cases this is often sufficient as the drying process always tends to break up the curd and make the milk more assimilable

Once again it is necessary to stress the point that parenteral infections will often simulate the simple dyspepsias and must be looked for in every case

### (3) Fat Dyspepsias

The percentage of fat as we have seen is approximately the same (3.5 per cent) in human and ordinary cow's milk. Certain types of cows (Kerry and Jersey) produce a milk with a very much higher fat percentage (6.5 per cent). The fat globule in human milk is much smaller than that of cow's milk and in some dried milks prepared by the roller process the fat globule tends to be larger still

Consequently it is not uncommon to meet a fat dyspepsia in perfectly normal babies who have been fed often quite unintentionally on some such high fat milk. Certain infants, particularly backward undeveloped babies sometimes show an idiosyncrasy even to the amount of fat present in ordinary milk

**Symptoms** Vomiting is a prominent symptom the vomit consisting of curdled sour smelling (smell due to butyric acid) milk often containing mucus. The vomiting does not usually occur immediately after the feed but some twenty to sixty minutes later. The bowels may be either constipated or loose. In the former case there will be colic and flatulence in the latter, loose large acid foul smelling stools grey white, or yellowish green in colour and always pale and slimy in appearance. Curds consisting of soaps may be seen in the motion and may be mistaken for protein curds by the inexperienced. If the stools are chemically analysed an excess of unsplitted fat will be found

**Treatment** *Prophylactic* Rich milks and the addition of cream are to be deprecated as a general rule in infant feeding. In the past the addition of cream was often advocated, but

now that it is possible to supply the necessary vitamins A and D in the form of cod liver oil or synthetic product this is no longer necessary. It is our general experience that children in Dublin do well on low rather than high fat mixtures. Not uncommonly particularly in the well to do farming community cases of fat dyspepsia are encountered due to an infant being given the milk from some special prize herd of Kerry cows.

*Curative* When the condition is diagnosed a dose of castor oil should be given and a stomach lavage performed if there has been much vomiting of mucus. The child should be starved for from twenty four to forty eight hours only being given water and sugar by mouth. At the end of this period he should be put on a skimmed milk or a half strength half cream dried milk with additional sugar added and then gradually put back on a normal milk mixture in which the percentage of fat is kept on the low side.

## B DYSPEPSIA DUE TO INFECTION

### (1) Parenteral

*Any infection occurring in any part of the body may cause an upset of the baby's digestion. The commonest of these parenteral infections are otitis media and naso pharyngitis bronchitis pyelitis and furunculosis are also occasionally associated with infantile dyspepsia. The symptom complex is extremely variable and all grades from acute diarrhoea and vomiting to occasional vomiting and loose stools are met with. About the commonest picture is that of a child who is brought to the doctor for ceasing suddenly to gain weight and going off his food. The mother says the child vomits once or twice during the day and that the stools have become loose some times green and sometimes yellow in colour. Sometimes she will volunteer that he is particularly fretful and that he has been crying and pulling at his ears. The temperature is found to be about 100° F. Examination of the patient reveals a red bulging drum in one ear and nothing else of note. Paracentesis is performed aural discharge follows and in a few days the child improves.*

It may be extremely difficult to find the source of infection. It is by no means a simple matter to see the drums of a small

infant even when an electric auriscope is used and the wax cleared away with an aural curette. The meatus may be very small wax may be tenacious and the pædiatrician's patience worn out trying to examine the screaming infant before a good view of the drums has been obtained. Again it is by no means a simple matter to obtain a specimen of urine from a small infant particularly the female baby.

It is impossible to lay too much stress on the importance of careful examination of every infant (such examination to include the ears and urine) who is brought to the doctor for any form of intestinal upset. Indeed it is often said that the commonest mistake in pædiatrics is that of failing to observe some parenteral infection underlying what appears to be a simple dyspepsia or an acute gastro enteritis.

**Treatment** Treatment is primarily that of the infective cause—the otitis pyelitis etc. and then that of the secondary gastro enteritis. No hard and fast rule can be made for the latter as every case is different. In one case no change of diet is necessary once the focus of infection has been dealt with—in another it is wise to reduce the strength of the feed till the infection has been got under. In others the gastric upset has been so profound that it will be necessary to starve the infant for twenty four to forty-eight hours and then recommence feeding with some simple easily digested formula as in the case of summer diarrhœa.

## (2) Enteric

Under this heading it is necessary to include a number of clinical and bacteriological entities such as gastro enteritis summer diarrhœa diarrhœa and vomiting of infants dysentery and cholera infantum. It is not possible here to review the literature on this subject except to state that bacteriologically gastro-enteritis may be divided into two main groups (A) In which one of the dysenteric organisms is associated with the condition, (B) in which they are not. No figures are available in this country as yet upon this subject and those from other countries are very variable.

The treatment of the condition is largely dietetic hence a full description is given in this chapter though from other points of view the subject might be better placed in that part of the book dealing with infectious disorders.

## GASTRO-ENTERITIS

Gastro enteritis occurs almost entirely in bottle fed babies during the fly season. In Dublin an epidemic of the disease occurs with perfect regularity about the end of the first or second week in July and continues for from six to eight weeks. In various investigations the fly has been definitely marked down as the carrier of the disease. In Dublin due to the number of cattle markets and the proximity of the bracken covered hills the summer fly plague is particularly severe and summer diarrhoea accounts for more deaths than any other cause during the first year of life.

The symptom complex is regular though the intensity of the symptoms is very variable. A healthy infant suddenly vomits a few hours later the motions become loose and diarrhoea commences. In some cases the vomiting is the most prominent feature in others the diarrhoea in some both are equally severe. The temperature may or may not be raised in the severer types particularly those associated with a definite dysenteric organism it may reach  $102^{\circ}\text{F}$ . In all cases there is some degree of dehydration due to loss of fluid by the bowel. In the dysenteric type particularly when the causal organism belongs to one of the virulent dysenteric groups, blood and mucus are found in the stools. In the worst cases severe dehydration collapse and death may occur within twelve hours of the onset. In other cases the condition is almost subacute and may drag on for days and even weeks. The more severe the diarrhoea the greater the loss of base from the bowel and the greater the resulting acidosis and dehydration will become.

**Treatment** *Prophylactic* Breast feeding during the dangerous season scrupulous attention to cleanliness of feeding utensils boiling of cow's milk and elimination of flies are the main headings here.

Active treatment is threefold —

- (a) Methods to combat the infection and intoxication
- (b) Methods to combat the dehydration
- (c) Methods of feeding so as to maintain the child's strength and at the same time supply him with a food which will not be vomited and will not aggravate the diarrhoea.

(a) **Methods to Combat the Infection and Intoxication**  
First of all every method of elimination must be used. In a

severe case where vomiting and diarrhoea are both prominent features the stomach should be washed out with a weak bicarbonate solution and a dose of castor oil (*Ol Ricini*  $\bar{3}$ i) given to the infant. Starvation should then be commenced and nothing but sugar (5 per cent) and water given by mouth (Do not give albumen water or barley water the former tends to make the child sensitive to eggs in later life and both are almost valueless from the caloric point of view). To begin with the diarrhoea should not be checked. Once the bowel has been cleared out completely however further purging should be avoided and attempts may be made to check the diarrhoea. *Ol Ricini* given in small doses has a constipating action and has been widely used in England. The well known *Mist Ol Ricini* (*Ol Ricini*  $\mathbb{M}$ v *Mucilage* q s *Aqua ad*  $\bar{3}$ i) may be given four hourly.

Kaolin is claimed by some to be of considerable value in absorbing the toxins formed in the bowel. The author has found the patent medicine Kaldrox (colloidal kaolin and 20 per cent aluminium hydroxide) of very definite value in a number of cases. When the infecting organism is of the dysenteric type and the large bowel is chiefly involved bismuth combined with opium may be of value (Dose *Tinc Opi*  $\mathbb{M}$  $\frac{1}{2}$  for an infant of six months and double this quantity at one year).

(b) *Methods to Combat Dehydration and Acidosis* Before dealing with the actual methods of treatment it is necessary to describe briefly the biochemical state of the blood. Diarrhoea leads to loss of fluid, loss of fluid produces a diminished blood volume. The concentrated blood is increased in viscosity and this leads to a sluggish peripheral circulation and decreased renal efficiency. The tissue oxidation becomes incomplete and certain acids are produced. A vicious circle is established the kidneys becoming further damaged by the toxæmia and so excreting less. All the while there is excessive loss of base from the bowel. Hence a condition of acidosis shown by a fall (often 50 per cent) of the alkaline reserve of the blood takes place.

Treatment therefore must be arranged so as to reduce the dehydration and acidosis as quickly as possible.

The usual method advocated is to give normal saline and glucose 5 per cent subcutaneously intraperitoneally or intravenously (fluid given by mouth is seldom of value in the acute

stages as it tends to be vomited or passed by the bowel almost at once) Recently it has been pointed out that the administration of normal saline is inclined still further to lower the alkaline reserve and increase the acidosis, and that it is better to give 0.45 per cent saline and glucose. It is stated that if this is done the fluid will be retained and the patient benefited at once.

The route used depends on the circumstances. If the administration has to be left to the nurse the subcutaneous route must be used and 4-6 oz. run in by gravity once or twice a day. If a doctor is available to carry out the treatment himself the intraperitoneal route is the easiest. Providing that careful aseptic precautions are used and the skin below the umbilicus picked up between the finger and thumb and the needle passed upwards through the abdominal wall there is no danger. Six ounces is about the best quantity to give by this method. It should be run in by gravity while the baby is quieted with a bottle of sugar and water.

The intravenous method requires experience, elaborate apparatus and careful supervision. An apparatus can be fixed up which enables (by a drip method) the administration to the child of a slow infusion into a vein for twenty-four to forty-eight hours. It is a most life-saving procedure in severe cases, but can only be employed in hospital by a trained staff.

Blood transfusion is advocated by some of the American school, but is contra-indicated in the author's opinion in cases of dehydration as it tends still further to overload the cellular elements in the circulation. A serum transfusion is however, often an exceedingly valuable therapeutic measure.

100-200 c.c. of blood is obtained from a suitable donor placed in a sterile retainer, covered with gauze and placed in the refrigerator over night. Next morning the clot is loosened and the serum decanted off and filtered. It is warmed to blood temperature and then 10 c.c. per lb. of body weight is given to the infant intravenously followed by double the quantity of saline and glucose. The fluid must be run in very slowly, the procedure taking not less than half an hour.

(c) Feeding Methods. The standard method is as follows. The infant is given nothing except water or water with glucose 5 per cent by mouth for twenty-four hours. Sometimes starvation has to be prolonged for forty-eight hours or longer if the condition does not respond at once. At the end of this

period feeding is commenced a whey mixture or half strength, half cream dried milk (such as half cream Cow and Gate) being used at first. The strength is gradually increased till the infant is getting a normal feed for its weight. As a rule it is necessary to continue with some simple mixture such as half cream Cow and Gate or half strength Trufood for some weeks till the digestion has completely returned to the normal. This simple method when combined with the other measures outlined above is sufficient to cure the majority of these patients. The very severe case however is extremely hard to treat and every measure may be unavailing. Certain cases fail to clear up and continue in a subacute condition for weeks or months. Many forms of feeding have been advocated for these some of which are outlined below.

**Acidified Milk Mixtures** The gastric acidity of infants with gastro enteritis is usually lowered and there are many advocates of lactic acid milk feeding for these cases. Such a milk can be prepared by adding 60 minims of lactic acid B.P. to one pint of boiled skimmed milk and half a pint of boiled water (the resulting mixture should have a pH 4 approximately). If sugar is to be added the best form to give it in is probably dextrin maltose. Another method is to ferment the milk for six to twelve hours with some lactic acid producing germ as *B. acidophilus*. This fermented (buttermilk) milk has a creamy consistency due to the very fine precipitation of casein curds.

**Protein Milk Feeding** Finkelstein first recommended this method as a way of treating acute diarrhoea in infants. It has had considerable vogue in America and in Germany but has never been used extensively here. The method of preparation is as follows —

A quart of whole milk is curdled with rennin. When firmly coagulated it is poured on two layers of cheese cloth and suspended for one hour so as to drain off the whey. The dry curd is rubbed through a vegetable masher with gradual addition of one pint of buttermilk. Enough boiled water is added to make one quart.

**Whey** Some advocate keeping the infant recovering from gastro-enteritis on whey for a week or longer before increasing the strength of the feed.

**Method of Preparation** Add two teaspoonfuls of rennet to 1½ pints of warm milk allow to cool. Now strain the curd through muslin and use the exudate. Secway (a Trufood product) is a dried whey powder and is a very useful preparation.

and one easily prepared. Whey has a very low protein content (8 per cent approximately) and practically no fat, but a normal percentage of sugar and salts.

**Peptonisation** This method of predigesting the milk is another good measure for helping the enfeebled digestion of infants in the convalescent state and may be continued for about four weeks (the method is described on p. 103).

### Dyspepsia Due to Anæmia

Anæmia (from whatever cause) is apt to be associated with digestive disorders though sometimes it is difficult to say whether the dyspepsia is the cause of the anæmia or *vice versa*. Undoubtedly however, correction of a marked anæmia in an infant often clears up what appears to be a chronic dyspepsia. Hence in all cases where anæmia may be suspected from the clinical appearance of the infant a blood examination should be done, and if an anæmia is found to be present it should be treated at once (see p. 189).

## CHAPTER XII

W R F COLLIS C L McDONOUGH

### VITAMINS

(*Vitamin D*—Rickets Pathology and Symptoms Bones of the Skull—Cranio-tabes—Ribs—The Extremities—The Pelvis—The Clavicle—Ligaments and Muscles—Dentition—The Blood—Infections—Tetany and Convulsions Late Rickets Renal Rickets—Celiac Rickets—Diagnosis Differential x-ray Diagnosis Congenital Syphilis Rickets and Scurvy Treatment. Hyper-vitaminosis D *Vitamin C*—Scurvy Signs and Symptoms—Diagnosis—Prognosis—Treatment *Vitamin B* *Vitamin A*—*Vitamin E*)

VITAMINS have been defined as food substances of no fuel value which are necessary to well being or life itself in the higher animals, but which as a rule they cannot manufacture for themselves

For centuries the necessity in diet for some of these factors has been known but it is only during the last twenty years that the problem has been elucidated and the disease syndromes resulting from vitamin deficiency worked out Even now much research work is needed for although the main defects of gross vitamin deficiency are known the effects of partial vitamin starvation are only beginning to be appreciated

The discovery of the principle of vitamins has revolutionised the subject of dietetics and is changing the whole conception of modern medicine for now we know that many perhaps more than half of the ills from which we suffer are due to faulty dietetics and that the commonest errors in diet are associated with vitamin deficiency The subject is of particular interest to the obstetrician and the paediatrician During pregnancy any deficiency in the mother's diet will be accentuated as she has to supply not only her own body with the necessary substances but also that of the fetus There is no doubt that many of the disasters of pregnancy are due to deficiency diseases and as time goes on we prophesy that more and more attention will be paid to the diet of the pregnant mother in the organisation of the ante natal clinics Again diet is a most important aspect of paediatrics give a child an ideal diet and he will have good teeth straight limbs and a strong body which will be able to

withstand infection. Neglect the now established principles and he will have carious teeth, a deformed and under developed body and be prone to catch every passing infection.

### Vitamin D ( $C_{28}H_{44}O$ , Irradiated Ergosterol)

Vitamin D is fat soluble and can be manufactured in the body by the action of sunlight upon the skin or taken by mouth. Its commonest sources in nature are certain animal fats *e.g.*, cod liver oil. Recently methods have been perfected for preparing it synthetically. It is one of the factors which control calcium metabolism and hence is particularly important in the pregnant woman and the child. Vitamin D controls the absorption of calcium from the alimentary tract and together with the hormone of the parathyroid glands which controls the deposition and mobilisation of calcium from the bones keeps the blood calcium at a constant level. Lack of vitamin D leads to osteomalacia in the adult and rickets in the child. Here we are only concerned with the latter.

Rickets, or the disease of darkness, is seen most commonly in the towns of the northern countries where the sun rarely shines and where its ultra violet rays are cut off by smoke. It occurs chiefly amongst the poorest sections of the community. Coloured children are more prone to the disease than white if they live in the northern cities due to the pigment of their skins. The disease is not confined however to any race or stratum of society but will appear anywhere if the necessary conditions for its occurrence are present. Rickets is most commonly seen between the ages of four and eighteen months of age. Occasionally it occurs earlier than the fourth month, a child of thirty four days with the disease was observed by Durham and recently reports of babies born with congenital rickets have come from China. It is one of the commonest diseases of early childhood being found in between 30-60 per cent of urban children. Active rickets is met with most frequently during the winter and early spring months *e.g.*, January-May. Weakly and premature infants are more prone to develop the disease than healthy full term babies.

**Pathology** Rickets affects primarily the bones and secondarily the muscles and ligaments. The amount of inorganic matter (calcium phosphate) is decreased in the bones and hence much of the supporting framework of the bone is lost and

it becomes weak and pliant. Rickets may affect almost every bone in the body, the shafts of the bones being decalcified (osteoporosis) and the epiphyses enlarged. The most characteristic lesion of the disease develops at the epiphyses of the bones. Here there is increased activity of bone formation and deficient completion of the process so that the epiphyses become enlarged and a line of ossification seen microscopically or by x ray (see plate) appears irregular and fluffy. In the flat bones the process is similar. In the skull the outer table becomes thinned and the inner table thickened. There is loss of supporting structure and if pressure is applied by the finger (particularly over the parietal bones) the bone may be indented like a membrane or parchment (craniotabes). When treated these rachitic lesions heal with great rapidity though in severe cases deformities remain.

(For fuller description of the pathology of rickets the reader is referred to the works of Howland and Kramer \* Park † and Shipley ‡)

**Symptoms The Bones** (a) *The Skull* *Craniotabes* is often the earliest symptom of the disease and is due to the development of the soft spots in the parietal bones (see above). They can sometimes be found as early as the third month. Bossing of the head particularly noticeable in the frontal bones develops as a rule during the second half of the first year. In severe cases the circumference of the head is increased by several inches and in these cases in spite of later healing the head often remains abnormally shaped: i.e. flattened on top and behind and prominent over the frontal and parietal bones. The fontanelle is late in closing and often remains open till after the second year and sometimes till the third.

(b) *The Ribs* The deformity of the chest in rachitic children is most characteristic. The ribs become beaded at the costo-chondral junctions—the rachitic rosary—the ribs become pliant and are sucked in during inspiration. Transverse grooves developing in the chest wall at the level of the diaphragmatic attachment—Harrison's sulcus—and the child becomes 'pigeon chested'. The liver and spleen may be pushed down and so become palpable. Considerable variation in the degree of deformity is found. In severe cases respiration becomes

\* Howland and Kramer *Amer Jour Dis Child* 1911 93: 107

† Park *Canad Med Assoc Journ* 1931 26 p 3

‡ Shipley, Kramer and Howland *Boston Journ* 1906 20 p 379

shallow and less efficient and secondarily respiratory infection often occurs. In milder cases the condition may pass unnoticed and deformity of the chest only be discovered in later life.

(c) *The Extremities* The most characteristic changes are enlargements of the epiphyses at the wrists and ankles. The epiphysis may tilt and give a false alignment to the bones, "knock knee" or "bow legs" resulting. The shafts of the long bones bend as the child begins to crawl or walk (e.g., bowing of the tibia and fibula, *coxa vara*, etc.)

(d) *The Pelvis* Rachitic changes in the pelvic bones result in a shortening of the antero-posterior diameter of the pelvis, which in women may lead later to obstructed labour.

(e) *The Clavicle* The clavicle is not uncommonly affected by rickets, showing an exaggerated anterior curve and enlarged extremities.

All the long bones become liable to fracture, which may be single or multiple, the trauma sometimes being so mild that the condition passes undiagnosed.

*Ligaments and Muscles* The ligaments become lax and elongated, the muscles lose tone, becoming flabby and poorly developed. Hence the spine is often affected in rickets, a marked posterior curve developing as a rule, though occasionally lateral curves are also seen. Looseness of the knee, hip, shoulder and wrist joints is also often observed, due to the same causes.

The rachitic child is always late in sitting up and standing. The abdomen becomes enlarged and the child is usually constipated.

*Dentition* The progress of dentition is almost always retarded. The appearance of the first teeth is later than usual and the whole process is retarded.

The recent work of Professor and Mrs. Mellanby on the part played by vitamin D in the formation and health of the teeth is of great importance. They have shown that lack of vitamin D before eruption of the teeth causes them to be ill formed with irregular structure (the enamel being deficient). This irregularity of the teeth allows dental caries later. On the other hand, if the infant has sufficient vitamin D while the teeth are undergoing formation their development is regular, their enamel complete and subsequent dental caries very much less likely to occur. They have also demonstrated that if the

structure of a tooth is later damaged the affected area will "heal" by the laying down of secondary dentine provided that the child has sufficient vitamin D in the diet at the time. A sufficiency of phosphorus and calcium is also necessary for good teeth formation. Cereals have a certain anti-vitamin D action and hence when groats or other cereals are added to the child's diet the amount of vitamin should be increased.

Hypervitaminosis D on the other hand will produce teeth excessively mineralised and rigidly fixed to the jaw bone.

**The Blood.** Anæmia is commonly found associated with rickets. It is not part of the rachitic process but rather due to the diet being deficient in iron or to intercurrent infection (see below).

**Infections.** Bronchitis gastro-enteritis stomatitis etc. are commonly found associated with rickets. It is probable that such infections are not due directly to lack of vitamin D but rather to lack of vitamin A and general debility.

**Tetany and Convulsions** may complicate rickets (the subject is fully dealt with on p. 125).

**Diagnosis.** Marled rickets can always be diagnosed without difficulty if the above symptoms are sought. Incipient rickets, particularly in the small infant, may cause considerable difficulty in diagnosis. As we have seen *craniotabes* is one of the earliest symptoms of the disease. It must not be confused with flexibility along the suture lines, a condition commonly met with in premature and weakly infants. The latter is not localised in patches as is rachitic *craniotabes* but may be felt along all the sutures and there is *no* crackling sensation when the bone is pressed upon by the finger. Occasionally, in premature babies and in *osteogenesis imperfecta*, much of the skull may be membranous but such conditions are extremely rare. In the older text books syphilis is often claimed as a cause of *craniotabes* but recent work suggests that it has only been observed in congenital syphilis when the latter condition was associated with rickets.

Beading of the ribs is another early sign. Here the condition must be differentiated from that found in scurvy (see p. 120).

Enlargement of epiphyses is a valuable sign but the diagnosis must not rest on this alone as enlarged epiphyses may be a familial characteristic. The enlarged bossed head must be differentiated from the general enlargement of the head in



CONGENITAL SYP. I



CONGENITAL SYP. II

hydrocephalus Most important of all, Pott's disease of the spine must not be mis diagnosed as rachitic curvature

If the diagnosis of rickets rested solely on clinical grounds it would be most uncertain, fortunately skiagraphs of the bones are of the greatest assistance to the physician, not only in regard to the general diagnosis of the disease, but also as indicating the degree of activity Usually for diagnostic purposes the lower end of the radius and ulna are used The first change to be seen is in the epiphyseal line which becomes irregular and indistinct In severe cases the epiphyseal end of the bone appears cupped and frayed out The shaft shows decreased density, due to osteoporosis and coarse trabeculation The stages of healing are also shown clearly in the skiagraph—calcification of metaphyses—lines of dense calcification, periosteal calcification etc Hence not only can the condition be diagnosed by x ray, but the whole process of healing followed and controlled Active rickets can usually be diagnosed without difficulty, but healing rickets may be confused with scurvy and considerable skill may be required in the x ray interpretation

### *Differential X-Ray Diagnosis of Congenital Syphilis Rickets and Scurvy*

Congenital syphilis in the infant gives rise to changes in the long bones which radiologically are diagnostic of the condition even when blood tests are negative

The characteristic appearances occur at the extremities of the shafts and consist of sub periosteal erosion of the growing portions of the diaphysis with a dense irregular metaphyseal line

Later ossifying periostitis may develop This might be confused with calcifying sub periosteal hematoma of scurvy, but can be distinguished by its coarser texture, which has been compared to a charcoal drawing while the appearance in scurvy resembles a fine pencil line (Plates V and VI)

A further distinguishing feature is the form of the shadow, which in scurvy is commonly more or less pear-shaped, due to the fact that most children suffering from the disease tend to lie on the back with the knees and hips flexed This position allows the extravasated blood to gravitate to the upper ends of the femora and the lower ends of the tibia and fibulae the

shadow stopping abruptly at the epiphyseal line. Since the periosteal thickening of syphilis is not affected by gravity it follows the contour of the bone.

Certain other conditions give rise to increased density of the metaphyses notably Albers Schönberg disease, lead poisoning and renal rickets but in all the sub periosteal erosion is absent.

Acute rickets produces a marked deformity, most easily demonstrated at the growing ends of the long bones and results in cupping of the diaphysis with ragged projections towards the epiphysis (Plates VII and VIII). The development of the epiphyses is nearly always but not invariably, retarded. Bending of the weight bearing bones is frequent.

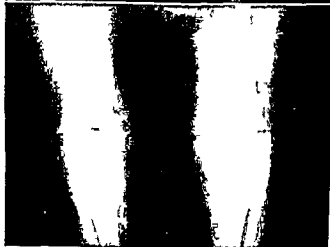
Scurvy, in addition to the periosteal changes already mentioned causes widening of the diaphyseal ends of the long bones with sclerosis and irregularity but cupping does not occur as in rickets.

### *Chemical Test for Rickets*

Howland showed that the most sensitive test for rickets is the estimation of the blood phosphorus and calcium. He states that if the product of these two substances expressed in milligrams per cent falls below 30 the child may be considered to be suffering from rickets.

**Treatment.** Rickets should never be seen once the principles of vitamin D deficiency are known. Vitamin D can be manufactured in the body by exposure to sunlight or taken by mouth. The amount of sunlight in the northern countries is hardly sufficient by itself to prevent the disease. Therefore it is wise to add some good source of vitamin D to the diet of all infants, children and pregnant and nursing mothers in these countries. The amount of vitamin D in unboiled milk varies and is often insufficient for the baby's needs. Hence we recommend giving cod liver oil from the third week of life onwards. At first half a teaspoonful a day is sufficient, this should be gradually increased till the baby is getting two to three teaspoonfuls by the end of the first year. If the cod liver oil is not tolerated one of the concentrated products such as adexolm, radiostoleum or halibut liver oil may be given instead—3 to 5 drops once a day being sufficient. Cod liver oil should be given uninterruptedly till after the second dentition. One of the commonest mistakes in all ranks of society is to cease giving cod liver oil when the weather is hot, or after the child

PLATE VII  
ACTIVE R KETS



HEALF R KETS

begins to take a mixed diet. The continued administration of cod liver oil or other source of vitamin D is of particular importance in regard to the formation of the teeth. Indeed if a healthy child receives sufficient vitamin D and milk up to the second dentition the permanent teeth will erupt in perfect condition and *will remain healthy afterwards*.

The treatment of active rickets consists essentially in supplying vitamin D and assuring that the child has a satisfactory milk supply. Vitamin D can be supplied by exposing the child to ultra violet light, or by administering cod liver oil or one of the vitamin D concentrates. In severe cases a combination of the two methods is sometimes recommended. If a severe case of rickets is met with it is wise to take the child off its feet while treatment is quickly instituted. When left untreated severe deformities may occur which will need surgical interference later.

### OTHER FORMS OF RICKETS

(a) Late Rickets. In children between seven and fourteen years who have been on a starvation diet a special form of late rickets may develop. The children complain of pains in the bones and joints. The former are thinned and bend and the epiphyses enlarge. The condition is rarely met with in Ireland.

(b) Renal Rickets. In certain children with chronic renal insufficiency a form of rickets has been described by Parsons. The bones show osteoporosis and deformity, and there is marked retardation of growth leading to the condition of renal dwarfism. The condition is due to alteration of the blood electrolytes—the phosphorus calcium ratio in the blood plasma being altered.

(c) Coeliac Rickets. Rickets occurring in association with coeliac disease is fully discussed on p. 137.

Hypervitaminosis. If vitamin D is given in too large doses (particularly the synthetic products) a condition of hypervitaminosis may supervene. The blood calcium becomes raised and calcification may occur in the artery walls and kidneys; the teeth may become hypercalcified into the jaw bones and fever and vomiting may occur. However, the danger of hypervitaminosis should the population become "vitamin conscious," is so small compared with the immense advantages that the abolition of rickets would confer upon the community that it can almost be discounted.

### Vitamin C (Ascorbic Acid $C_6H_8O_6$ )

This vitamin which is found widely distributed throughout nature in fresh fruits particularly oranges and lemons and root vegetablees such as the turnip and the potato is essential to life. Its exact mode of action is not known though recent work suggests that its presence is necessary for the essential process of tissue oxidation.

Deficiency of vitamin C at any age leads to the clinical syndrome of scurvy. Before the actual symptoms of scurvy appear the patient is in a subscorbic condition. Recent work with ascorbic acid has shown that for perfect health the tissues of the body must be saturated with vitamin C.

### Scurvy

Breast fed babies do not suffer from scurvy. In the artificially fed it is seldom seen before the fourth or the fifth month. The commonest age period in the child is between six and fourteen months. The disease is brought about in these children by a sterilised diet e.g. boiled or dried milk without the addition of orange juice or other source of vitamin C.

**Signs and Symptoms** The onset is gradual associated with pallor and fretfulness. The characteristic pathological lesion of scurvy is hæmorrhage. This may take place in the gums around the erupting teeth under the skin in the form of purpura from the kidneys giving rise to hæmaturia (an early sign) or into the epiphyses causing separation. The latter often leads to a backward displacement of the sternum and costal cartilages. If the finger is passed along the ribs it will suddenly fall over the edge when it reaches the costo-chondral junction due to the backward dislocation of the cartilage on the bone. This can be distinguished from the beading of the ribs in rickets where the finger is forced outwards by the enlargement of the epiphyses when it reaches the junction.

Sub periosteal hæmorrhages lead to the most characteristic symptoms of the disease. They are painful and hence the child is brought to the doctor for crying on being touched. On examination he lies on his back with the limbs externally rotated and screams if they are moved. Skiagraphs reveal characteristic appearances the sub periosteal hæmorrhages

PLATE VIII



RICKETS  
*Showing deformity due to weight bearing*

appear as shadows along the shafts of the long bones while a characteristic 'line' is seen just beyond the epiphyses due to diminished activity of the osteoblasts. An anaemia independent of that secondary to the haemorrhage is a feature of scurvy. No improvement can be effected in this anaemia till the scorbutic condition is treated. The administration of iron alone is useless.

**Diagnosis** The diagnosis is simple in cases of frank scurvy with definite haemorrhages but may be difficult in early cases. In the latter the finding of red blood corpuscles in the urine is an invaluable sign and the diagnosis can be made if the characteristic scorbutic beading of the ribs is found.

Acute anterior poliomyelitis may sometimes be mistaken for scurvy during its acute painful phase when the paralysed limb is tender. However, careful examination of the extremity in scurvy shows that the limb is not paralysed but held immobile so as to avoid the pain of movement.

Osteomyelitis has sometimes been confused with scurvy but the temperature of the child (e.g. normal in scurvy, high in osteomyelitis) and the swelling and pain which in osteomyelitis are confined to one limb only usually make the correct diagnosis possible while an x ray will settle the matter.

The epiphysitis of congenital syphilis is another condition sometimes confused with scurvy. Other signs of syphilis are usually present, a raised temperature is common and the x ray appearances are diagnostic (see p. 117).

**Prognosis** Once diagnosis has been made and treatment instituted the symptoms usually clear up with great rapidity. Patients suffering from scurvy are very prone to catch intercurrent infection and to die if they do so.

**Treatment** Scurvy should never be allowed to occur as it can be prevented by the simple procedure of giving the infant one to three teaspoonfuls of orange juice a day (depending on its age). Ample supplies of vitamin C are also contained in all fresh fruits, potato and turnip.

The most effective way to treat active scurvy is by the administration of ascorbic acid. This is usually followed by the subsidence of all symptoms in a few hours. If this drug is not to be had almost as satisfactory results can be obtained with large doses of orange or lemon juice.

Recent investigations upon the amounts of ascorbic acid found in urine suggest that the patient reaches the sub

scurbutic state a considerable time before the actual symptoms of scurvy appear. It is wise therefore, to start the administration of orange juice as early as possible in artificially fed babies. It used to be thought as scurvy was rarely seen before the fifth month that the third month was early enough for the introduction of orange juice. Recently we have introduced it about the end of the third week as a routine and are of the opinion that if the subscorbutic state is to be avoided orange juice administration should not be delayed beyond this date.

Recently ascorbic acid has been used in other hæmorrhagic states (e.g. thrombocytopenia) with success and we may hope for further developments along these lines in future.

### Vitamin B ( $C_{12}H_{16}N_4OS$ )

Vitamin B is another essential food substance whose deficiency leads to disease syndromes. It is a very complex substance which is found in germinating cereals, yeasts, fruits, vegetables, eggs and the liver, heart and kidneys of animals. Recent work suggests that the vitamin may be divided into two or more subdivisions. It has been shown that gross lack of  $B_1$  leads to beri beri (a disease of the nervous system associated with muscular inco-ordination and paralysis seen in the East amongst peoples whose staple diet is polished rice). Gross lack of  $B_2$  is said to be the cause of pellagra in maize eaters.

Apart from these main syndromes it has been claimed that vitamin B plays an essential part in hæmopoiesis (Parsons) that its administration is beneficial in certain infected skin conditions such as furunculosis and a curious form of syncope in babies born of mothers suffering from beri beri has been described by Bray amongst the inhabitants of certain Pacific Islands. Hence although absolute vitamin B deficiency is almost unknown in this country, slighter degrees of the condition may well exist especially amongst those pregnant women whose diet consists largely of white bread, tea and margarine and later amongst their infants.

Preventive treatment consists in assuring some source of vitamin B in the diet of the population. This could be done best by insisting upon the introduction of whole meal bread instead of white bread.

When a condition of vitamin B deficiency is found (or

thought) to exist, it can be made good by the introduction of eggs, yeast, Marmite, Gve, Virex or Beamax (wheat germ) to the diet

### Vitamin A ( $C_{20}H_{26}H\ OH$ )

The precursor of vitamin A is carotene ( $C_{20}H_{28}$ ) a pigmented substance found widely distributed in nature (e.g., in carrots, turnips, etc.) In the animal body it is synthesised into vitamin A and there stored, largely in the liver. Therefore man can obtain the actual vitamin itself from liver oils and animal fats or its precursor from carrots and green vegetables. Sufficiency of vitamin A is essential for the health of the body membranes. Lack of it leads to their devitalisation and infection. It was thought that vitamin A might be a definite anti-infective factor and was used by Mellanby and others in puerperal sepsis. The latest work does not bear this out, however, and the present position is somewhat as follows—if there is a vitamin A deficiency, infection is more likely to occur as a secondary phenomenon, but the administration of vitamin A in large doses to normal individuals will not protect them from pyogenic infections.

Complete lack of vitamin A produces xerophthalmia and other local infections. A very large proportion of blindness in India and China is due to xerophthalmia in infants and children. It is seldom, if ever seen in Ireland. In some of the distressed industrial areas in the north of England some cases of night blindness, which is the first symptom of the deficiency in the adult, have been reported recently. (Night blindness is due to lack of visual purple in the retina. Vitamin A seems necessary for its formation.)

Vitamin A is of real importance in paediatrics here, as minor degrees of its deficiency are not uncommon among artificially fed infants, the condition being characterised by a tendency to infection of the mucous membranes, e.g., gastro-enteritis, stomatitis, and respiratory disease etc.

Recent work suggests that vitamin A deficiency may play a rôle in the development of pyorrhoea in later life, but further investigations must be undertaken before this hypothesis can be accepted.

Prevention of any degree of vitamin A deficiency is a simple matter in this country once these principles are appreciated. Every pregnant mother should have a satisfactory supply of

green vegetables and all infants should be given cod liver oil which contains both vitamins A and D. The prevention and treatment of xerophthalmia in the East is outside the scope of this book.

### Vitamin E

For completeness a short reference must be made to the latest arrival in the vitamin field. This vitamin is concerned with sexual health, its lack producing sterility and death of the fœtus in utero. The experiments which have been carried out on rats are quite convincing but so far their application to man has not been fully worked out. It is a field of very great potential interest to the obstetrician and pædiatrician. The vitamin is found in wheat germ, maize germ, watercress and lettuce.

Finally it is necessary to remind the reader that this chapter is part of the dietetic section of a book concerned primarily with pædiatrics. Hence emphasis has been laid throughout on the preventive aspect of the problem rather than the disease syndromes which result from the different deficiencies. Secondly the subject is not static and therefore it has been possible only to outline the already known facts and point towards probable future advances.

## CHAPTER XIII

W J E JESSOP AND W R F COLLIS

### TETANY AND CONVULSIONS

(Tetany Signs Symptoms, Calcium Metabolism Vitamin D and Parathormone—Treatment Convulsions Causes, Diagnosis, Treatment)

THE term tetany is applied to a well defined clinical entity in which there is increased excitability of motor nerves and certain parts of the central nervous system. The condition varies in severity and in the milder cases, termed latent tetany, increased excitability is only demonstrated by the application of certain stimuli. The two most commonly used in diagnosis are tapping of the facial nerve anterior to the external auditory meatus, which produces spasm of the facial muscles on that side—Chvostek's sign—and application of pressure to the arm when the spasm produced causes the hand to assume the "obstetrical position"—Trousseau's sign. In more severe cases, sometimes called manifest tetany, spasms occur spontaneously or are evoked by such slight stimuli as are ordinarily unavoidable, such as contact with clothes, etc. The spasms are both tonic and clonic. The most obvious tonic spasms are those of the hands and feet—carpopedal. The hands remain in the "obstetrical position" for prolonged periods and the feet show a tonic plantar flexion at the ankle metatarso phalangeal, and inter phalangeal joints. The spasm may last for a few minutes or hours. A tonic spasm of the laryngeal muscles—laryngismus stridulus—prevents air from entering the chest. The patient becomes cyanosed, and when at last air is allowed to enter a "crowing" sound is produced. When this is very severe it may cause death by asphyxiation but it may be so mild that the sound can only be detected during forced inspiration as when a child cries (see p 428). Again there may be slight tonic spasm of the facial muscles with an immobile expressionless countenance.

Clonic spasms constitute the convulsions which are so serious a feature of the condition. They are most commonly generalised and vary in duration from a few seconds to several minutes.

They vary in frequency from perhaps once a week to an almost unbroken succession. They are generally accompanied by one or more of the tonic spasms but may occur alone. Animal experiment seems to indicate that they are produced by a hyper-excitability of the brain stem.

It is thus apparent that the parts of the nervous system affected are the motor nerves, spinal cord, brain stem and certain aspects of the autonomic system—in addition to the muscles of the larynx the œsophagus and cardia have been found to become spastic in some animals. It is of course not possible to elicit sensory symptoms like tingling and numbness in infants.

The immediate cause of hyper excitability is probably two fold. (A) *a deficiency of ionised calcium in the blood plasma* and (B) *a reduction of H ion concentration so that the blood becomes too alkaline*. These may be related to each other but it is best to consider them separately for the present.

(A) Calcium is taken into the body in certain foods notably milk and eggs. Only a comparatively small fraction of ingested calcium is absorbed, the remainder passes through the intestinal tract into the fæces. Calcium is excreted both by the kidney and large intestine. In order to preserve equilibrium the total ingested must not be less than the total present in urine and fæces and in children the ingested calcium should be in considerable excess. The absorption of calcium is influenced by a number of factors. If the intestinal contents are too alkaline insoluble carbonates and phosphates of calcium are formed and pass unabsorbed while the presence of mineral acids especially HCl tend to keep the calcium in solution and so promote absorption. Fatty acids form insoluble calcium soaps and so hinder absorption. *Vitamin D* is a most important agent for promoting absorption of ingested calcium, though the exact mechanism of its action is not understood.

The calcium of blood is derived from two sources. That absorbed from the intestinal tract as indicated above is supplemented by an extremely important fraction from the bones. Similarly calcium disappears from blood not only through the excretory channels mentioned above but also by deposition in bones. Consequently an important factor in regulation of blood calcium will be the relative activity of deposition on or absorption from bone. This is governed by *the secretion of the parathyroid glands*.

In their normal state of activity a balance is maintained so that the bones are not reduced in strength and the blood calcium remains at a value of 9-11 mg per cent. If the parathyroids become hyper active excess calcium is mobilised from bones and the blood calcium rises but if the parathyroid hormone is deficient blood calcium immediately falls even in spite of adequate intake. There is no evidence in such cases to show that absorption from the intestine is interfered with or that there is any increase in excretion so the amount deposited in bones must be increased. However as the tetany which results from reduction of blood calcium is so serious a condition that it must be corrected immediately it has never been possible to demonstrate any increase in calcium content of bones in such cases.

So far reference has only been made to blood calcium as a whole in point of fact nervous excitability probably depends only on the value of the ionised fraction. Of the total calcium present in blood about half is diffusible and of this fraction some is ionised and some non ionised. Total calcium may be determined with ease in serum or citrated plasma. The estimation of diffusible calcium is possible though difficult but the ionised fraction cannot at present be determined. On general principles however it is probable that ionised calcium is more closely related to diffusible than to non-diffusible or total values. It has been suggested that the calcium of cerebrospinal fluid represents the diffusible fraction of serum calcium and that its level might be more closely related to neuro muscular excitability than that of calcium in serum. But recent work by McCance Carmichael and others has tended to cast a doubt on the theory that the CSF is merely an ultra filtrate of plasma and McCance finds that its calcium content is determined to some extent at least by its protein content. In view of this uncertainty of the relation of CSF calcium to serum calcium attention will be directed in the remainder of this discussion to total serum calcium values unless otherwise stated.

From a consideration of the above account of calcium metabolism it will be obvious that the following factors may produce tetany by lowering blood calcium —

- (1) Extirpation or deficient function of parathyroid glands
- (2) Deficiency of calcium in diet
- (3) Failure of absorption of calcium from intestinal tract

The action of factors (2) and (3) may of course be counter balanced by increased parathyroid activity as we shall see presently

(1) Parathyroid tetany The most common cause is removal of the parathyroid glands at operation but occasionally a case of spontaneous onset will be found on post mortem examination to show hæmorrhages into the parathyroid glands. If very severe the condition may be relieved temporarily by intravenous injection of calcium chloride. More permanent benefit may be derived from injection of extract of parathyroid (parathormone). If this is maintained for some weeks the nervous system will become adjusted to the lowered blood calcium so that spasms may not supervene when treatment is discontinued.

(2) A deficiency of calcium in the diet is itself not a very common cause of tetany though it may be associated with deficiency of vitamin D. It is mentioned for the sake of completeness but its features are essentially the same as those of incomplete absorption and will be considered with them.

(3) Incomplete absorption of calcium is considered by most authorities to be associated with rickets and it is a recognised fact that the great majority of cases of infantile tetany occur in rachitic children. The cause of incomplete absorption may in certain instances be due to excess fatty acid in the intestine *e.g.* celiac disease but in nearly all cases it is deficiency in vitamin D. Guild has noted a lack of parallelism between the severity of the rickets and the tetany. In one series of cases of tetany quoted by her 58 per cent had only very mild rickets while only 6 per cent showed really severe bony changes radiologically. In the light of the above discussion it would appear that in one type of case the tendency to lowering of blood calcium produced by a low calcium absorption is counteracted by mobilisation of calcium from bones. Tetany is thus prevented but the bones suffer correspondingly. In the other type with less active parathyroids calcium is not mobilised from bones and blood calcium falls. Tetany will develop but the bones will not be so severely damaged. Since the principal function of the parathyroids is probably to maintain the level of blood calcium tetany produced by a low calcium absorption is to be regarded as a sign of relative failure of parathyroid activity under strain.

Tetany produced by deficiency in calcium absorption may therefore be treated in one of three ways. Calcium may be

added directly by intravenous injection of calcium chloride solution or by intramuscular injection of gluconate, or calcium may be mobilised from the bones by injection of parathormone. Both these may be used in severe cases as emergency measures, but neither is suitable for prolonged use. The latter would be particularly harmful as the blood calcium would be raised only at the expense of bony tissue. The rational treatment is obviously the correction of the cause of incomplete absorption. In rickets adequate supplies of vitamin D and ultra violet radiation are indicated while in coeliac disease the removal of fat from the diet will reduce the fatty acid content of the bowel. The danger of thus producing a deficiency of fat soluble D may be guarded against by use of one of the vitamin concentrates.

### Tetany produced by an alteration of Acid-base Balance towards the Alkaline Side (Alkalosis)

If the blood pH is raised above 7.7 the individual generally develops tetany. The tetany produced in this way is not related to a lowering of total serum calcium. There are two possible explanations. Reduction of H ion concentration may reduce the concentration of ionised calcium without altering the total calcium, or there may be a direct increase in excitability of the nervous system by increased blood alkalinity. Examples of the condition are seen in cases of excessive alkali intake, as in the alkaline treatment of nephritis, in cases of pyloric obstruction, where loss of chloride in the vomit will leave an excess of basic radicals in the blood stream, and following excessive pulmonary ventilation, either voluntary or in conditions like encephalitis lethargica, the loss of  $\text{CO}_2$  again leaving an excess of basic radicals.

The tetany of alkalosis may be relieved by raising the H ion concentration of the blood as by administration of ammonium chloride.

### CONVULSIONS

Convulsions due to birth trauma have already been described. Here we are concerned only with convulsions occurring after the neo natal period particularly between the ages of 6 and 18 months. Convulsions occurring suddenly without warning are

a common occurrence in this latter age period. They are seen in all grades of society and are associated with a large number of pathological states which may be difficult to diagnose.

(1) Tetany complicating rickets is probably the commonest cause of convulsions at this age period (see above).

(2) Acute infections, *e.g.* pyelitis measles whooping cough, etc. are not uncommonly ushered in by a fit.

(3) Acute cerebral conditions (*e.g.* meningitis) both in the early and late stages are often associated with convulsions.

(4) Epileptic fits may commence at this age.

(5) Poisons both bacterial and other may cause convulsions *e.g.* tetanus rabies strychnine or insulin (hypoglycemia).

Sometimes fits occur and no cause can be found to account for them. These are termed idiopathic and are supposed to be associated with such conditions as teething or constipation in the child. Recent work has shown however that often such convulsions are in fact caused by tetany.

**Diagnosis.** The problem of diagnosis of the cause of convulsions is often one of extreme difficulty and may be impossible when the doctor is first called to the case particularly in a private house where the parents may be in great distress. The fit having been controlled (see Treatment) the baby should be carefully examined. First the temperature should be taken and the child examined for any signs of infectious disease. If there are no signs of the latter and if the temperature is raised and there are any signs of meningitis such as stiffness of the neck or Kernig's sign a lumbar puncture should be done forthwith and the fluid examined. Lumbar puncture appears to have a beneficial effect in most cases of convulsions and will do no harm in any case. Hence if there is any doubt in the doctor's mind it should always be done. The temperature may be raised in tetanus but the condition is hardly likely to cause confusion, constant trismus will be found and the child's appearance is characteristic (*e.g.* the risus sardonicus etc.). Strychnine poisoning is occasionally seen in babies who have an idiosyncrasy towards the drug and convulsions have been known to occur in such cases. The convulsions resemble those seen in tetanus but relaxation occurs between fits and the temperature of the child is usually normal.

In cases where these acute conditions have been ruled out, the diagnosis will lie between tetany, epilepsy and idiopathic

convulsions In a typical case of tetany facial irritability (Chvostek's sign), Trousseau's sign and the general appearance of the fit as described on p 125, together with coincident rickets will make the diagnosis obvious Often, however, these special signs are absent for some time after a convulsion, and the fit itself may not have been typical In these cases the diagnosis can only be made by an estimation of the blood calcium a procedure which takes time, and is not always possible Under such circumstances if the child shows signs of rickets it is well to assume that the case is one of tetany, and treat it accordingly

If fits recur at regular intervals, in spite of treatment, careful attention should be paid to the child's general health, and, in the absence of acute infection epilepsy must be borne in mind though it is wise not to make the diagnosis till the infant has been under observation for a considerable period of time

**Treatment** *Preventive Treatment* Convulsions will not occur in healthy children who have been fed on a balanced diet Prevention of rickets means prevention of tetany and hence our readers are referred to the section on dietetics for further details of prophylactic treatment

*Treatment of the Fit* First it is necessary to reassure the parents Babies very rarely die in convulsions but parents are always in a state of extreme alarm on these occasions Therefore on entering the house the doctor's first duty is to reassure the mother and father and then confidently proceed to control the fit First counter irritation should be applied The old remedy of a hot mustard bath is satisfactory, though it is simpler and usually sufficient to place the baby's legs in hot water and apply cold to the head The usual fit is self limited, but it is wise to do something and hence the above measures should always be taken

Washing out the bowel and leaving in a rectal injection of chloral hydrate is of little value If the fit continues in spite of counter irritation, it is best to apply ether anaesthesia While the child is anaesthetised a lumbar puncture should be done both for the purpose of ruling out meningitis and so as to reduce the pressure of the cerebrospinal fluid If tetany is diagnosed or suspected, treatment should be commenced at once The convulsions of tetany can be controlled instantly by the inhalation of 30 per cent carbon dioxide in oxygen If this

is not to hand light ether anaesthesia may be used. As we have seen, the immediate cause of these convulsions is a diminution in blood calcium the administration of calcium salt is therefore indicated. Calcium chloride is found to be the most efficacious and should be given in ample doses. This salt is irritating if given subcutaneously or intramuscularly and hence should be administered intravenously or by mouth—three doses gr  $\text{xx}$  in milk by mouth at three hourly intervals can be given to a child of six to nine months. At the same time vitamin D in a concentrated form (e.g. radiostoleum adexolin etc.) should be given and the child's diet regulated. Small doses of sodium luminal (gr  $\frac{1}{2}$ – $\frac{1}{4}$  b.d.) or chloral hydrate (gr  $\frac{1}{2}$ –1 t.i.d.) to an infant of six months should be given for the next few days till the general anti-rachitic treatment has had time to take effect. Another method of raising the blood calcium rapidly is to give the child an injection of parathormone. This is a very satisfactory way of controlling the convulsions of tetany. When used it should be accompanied by an intramuscular injection of calcium gluconate (20 c.c.) and followed by the anti-rachitic treatment described above.

The treatment of the convulsion in cases of meningitis or other acute infection is that of the primary condition. If epilepsy is diagnosed sedatives must be commenced forthwith. Sodium luminal is usually the best drug for infants and young children with epilepsy. It is always wise to commence with small doses such as sodium luminal gr  $\frac{1}{2}$  b.d. and to increase slowly till the desired effect is obtained.

In those cases where no cause is discovered for the convulsion the child should be kept under observation for some time and if nervous given small doses of sedative for some weeks while the diet, bowels and general condition are regulated carefully.

## CHAPTER XIV

R F STEEN

### CÆLIAC DISEASE

(Historical—Ætiology—Pathology—Symptoms Enlarged Abdomen Wasting Stools Weight Chart Precociousness Slight Pyrexia Secondary Anæmia Flat Sugar Tolerance Curve—Complications Bronchitis and Pneumonia Œdema Bed sores Rickets Tetany Scurvy—Diagnosis Fat Indigestion in Infants Chronic Intestinal Indigestion Tuberculous Peritonitis Rickets—Prognosis—Treatment Diet Vitamins Drugs Nursing)

**SYNONYMS** intestinal infantilism Gee's disease

Cœliac disease has aptly received its name since the word *cœliac* is derived from the Greek word *κοιλια* a belly and enlargement of the abdomen is one of the most striking features of the disease

**Historical** Samuel Gee (1839 1910) first described it in 1888 Later Cheadle gave it the title of *acholia* and Herter in America *intestinal infantilism* Later again Heubner described it in Germany and on the Continent it is referred to as Heubner Herter or Gee Herter's disease If any name should be attached it should be that of Gee to whose original description little has since been added

**Ætiology** The cause is obscure Any theory as to explain the large amount of faecal fat which is for the most part of the split (i.e. digested) type

*Various theories have been put forward —*

(1) At one time it was thought to be due to *insufficiency of the pancreatic and biliary juices* This view is incorrect because (a) the duodenal contents have been examined in cases of cœliac disease and no evidence of any such deficiency has been found (b) the fat in the faeces is of the split type i.e. most of it is in the form of fatty acid indicating that its digestion is not at fault

(2) Herter's view was that it was caused by a *specific microbe* but against this is the fact that no such microbe has with any constancy been found in the faeces and though occasional rises of temperature are common enough throughout the disease

they are so slight and at such infrequent intervals as to discount an infective origin

(3) Leonard Parsons considers that the probable explanation of coeliac disease lies in *a change of a physico chemical nature in the absorptive mechanism of the intestine*. What causes this defect remains unsolved. The close resemblance between coeliac disease and tropical sprue would in the author's opinion lead one to hope that any discoveries made with regard to sprue might have an important bearing on coeliac disease.

**Sex Incidence** Both sexes are affected girls slightly more often than boys

**Age Incidence** In this respect coeliac disease resembles a large number of other diseases of childhood in being confined to a narrow age incidence. It is rarely seen before the first year of life the youngest case in the author's experience being eleven months old. The condition tends to undergo spontaneous cure about the fifth year but by treatment this can be effected much earlier. Cases are described in later childhood but these are so exceptional that one should hesitate to make a diagnosis except on very strong evidence.

**Pathology** Nothing characteristic is found at post mortem. The intestine is dilated and its mucous membrane atrophied but this would be expected in any condition with such extreme wasting.

**Symptoms** (1) *Enlarged Abdomen* This is in marked contrast to the wasted condition of the rest of the child (see Fig. 13) and makes the description 'the belly of a poisoned pup' which has been given to the abdomen of rickets even more apt in the case of coeliac disease and is the feature which makes the disease very liable to be mistaken for tuberculous peritonitis. The distension in coeliac disease is due to flatus not fluid though a small degree of ascites is said to occur occasionally in severe cases.

The chronic meteorism appears to be due to two things —

(a) Accumulation of gas in the intestine from fermentation

(b) Weakness of the musculature of the bowel and abdominal wall

Even when with treatment the stools improve and the patient appears recovered from the disease the abdominal distension persists for a long time. It is important however to remember that during an attack of diarrhoea or during those

"crises" which occur in the weight chart, the abdomen may become collapsed

(2) *Wasting* The contrast between the large abdomen and the wasted condition of the rest of the child's body is very striking. In no other disease can such extreme wasting occur and death appear imminent and yet recovery take place. In one case under the author's care the child at one year and four months old was only 7 lb 14 oz i.e. the birth weight of a normal child, and yet she recovered. The wasting is particularly marked in the buttocks which present a characteristic flat and wrinkled (*vide* Fig. 13) appearance. The face is



FIG. 13.—Celiac disease showing enlarged abdomen, wasted limbs, and lack of subcutaneous fat.

usually less wasted in comparison to the rest of the body, and often belies the appearance of the child when it is stripped.

(3) *Characteristic Stools* The macroscopic appearance of the stools is highly diagnostic. They are bulky, pale, lumpy, greasy and very offensive, and may be likened to lumpy porridge. Microscopically fatty acid crystals are usually present in excess. Chemically, the faecal fat is found to be enormously increased. The ratio of "split" to 'unsplit' fat is similar to that found in the normal stool, showing that the fat in its passage through the bowel has been digested perfectly and that the failure is one of *absorption* rather than of digestion. Normally the faecal fat forms less than 25 per cent. of the dried faeces. In celiac disease it is usually found to be 50, 60 or even 80 per cent. Too much stress, however, should not be laid on the percentage

increase in the faecal fat as a diagnostic feature since some increase tends to be associated with any form of diarrhoea in children *e.g.* tuberculous peritonitis. A normal percentage figure rules out coeliac disease but a raised figure does not necessarily indicate that the disease is present unless the figure is a very high one (in the region of 60 per cent). When doubt exists Leonard Parsons recommends confirming the diagnosis by estimating the total *output* of fat *per diem*. He considers that the stools in coeliac disease always contain more than 2 gm. of fat daily. The figure is usually higher and may reach 20 gm. or even more. In making such estimations the faeces are collected over a period of several days and the average taken as the weight of the stools varies a great deal from day to day.

(4) *Weight Chart* This is very characteristic in coeliac disease. A remarkable feature is the constant level which the weight may show for weeks months or even years. The author knows of one case where the weight remained stationary at 12 lb. for over two years. Fluctuations of course occur and indeed such fluctuations are very characteristic of the disease but the general level remains constant. A very frequent feature is a gradual gain in weight of 1 or 2 oz. *per diem* for perhaps a week or ten days and then within twenty four hours there is a *crisis* and the weight drops to its original level with a loss of perhaps a pound or more. Another feature sometimes seen is a *swinging* type of weight, so that 2 or 3 lb. may be gained in one day and lost the next, this is due probably to water retention and loss. It is essential that the mother and doctor should realise that such daily alterations in the weight are no index at all of improvement or the reverse. It is only the general trend over several weeks or months that matters. Accompanying the relatively stationary weight is a cessation of *growth* hence the name 'intestinal infantilism'.

(5) *Precociousness* This is often mentioned as a very characteristic feature. Speech it is true is often markedly delayed even to the extent of mutism but the child on the other hand is extremely advanced in his ability to understand what is going on around. He takes a great interest in the diet and tends to display the mentality of the chronic invalid of later life. This mental picture is probably not in any way due to the disease *per se*, but is an outcome of the prolonged of the illness.

(6) *Other Features* (a) A slight pyrexia is common from time to time, but is not in any way characteristic. It may be due to toxic absorption from the bowel excitement, or some other simple and transient cause but it is not of a type to suggest in the least that it is a part of the disease syndrome.

(b) A secondary anemia of low colour index (hypochromic type) is met with similar to the nutritional anemia of infancy. Much less commonly an anemia of megalocytic type is found similar to pernicious anemia. This is due probably to failure of absorption of the substance resulting from the interaction of Castle's intrinsic and extrinsic factors and in these rare cases parenteral liver extracts (e.g. campolon) should be given.

(c) A flat sugar tolerance curve is usually encountered but is not sufficiently characteristic to be of any great diagnostic value.

*Complications* These may be classified into two groups —

(1) Those due to the debilitated state of the child

(2) Those due to vitamin deprivation

With the improvement in modern treatment the latter should become rarer.

(1) *Complications Due to the Debilitated State of the Child*

(a) *Bronchitis and Broncho pneumonia* All the respiratory diseases are apt to be associated with celiac disease.

(b) *Edema* This is probably of the nutritional type similar to that described in 'Conditions Due to Starvation'.

(c) *Bed sores and Gangrene* These must be guarded against. If salines are being given subcutaneously great care should be taken that asepsis is observed.

(2) *Complications Due to Vitamin Deprivation* (a) *Rickets*

When one considers the excessive loss in the stools of calcium (as calcium soaps) and the failure of absorption of vitamin D which must accompany the lowered absorption of fat it is not surprising that rickets is a common complication of the disease. The treatment with a fat free diet where this is not sufficiently supplemented by one of the vitamin concentrates is an additional cause of rickets. However even when deprivation of vitamin D would seem to be almost complete, the bones do not show the characteristic changes in the epiphyses, but only a marked degree of osteoporosis. This is due to the fact that in the active stage of celiac disease growth tends to cease. "Celiac rickets" in its florid form tends to develop in the stage of recovery, i.e., in the age period four to seven years.

when growth is usually beginning to take place with great rapidity and when as Harrison has shown even the normal child shows exaggerated growth. This is a time when if a fat free diet is continued the vitamin content should be carefully attended to in order to make sure that it is adequate and if necessary vitamins by mouth may be supplemented by ultra violet therapy.

(b) *Tetany* This is a manifestation of rickets and is due to the vitamin D deficiency causing a hypocalcæmia.

(c) *Infantile Scurvy* This is due to vitamin C deficiency. If fruit juice is included in the diet this complication should never occur.

**Differential Diagnosis** (1) *Fat Indigestion in Infants* When the diet is too rich in fat e.g. Jersey cows milk full cream dried milks etc. infants particularly in the summer months may develop pale loose stools or diarrhoea with an increased percentage of fat in the faeces. This may be distinguished from coeliac disease by the fact that the latter is rarely seen in infancy and certainly never in the first six months and by the fact that simple fat indigestion responds to a reduction in the fat content of the diet whereas coeliac disease may persist for months or years in spite of the most careful dieting.

(2) *Chronic Intestinal Indigestion* (chronic intestinal dyspepsia chronic gastro intestinal catarrh mucous disease). This is the name given to a condition found at a later period than infancy where the child presents symptoms of indigestion with irregularity of the stools usually caused by irregular meals associated with the excessive consumption of sweets starchy foods etc. It is distinguished from coeliac disease by the fact that it usually responds quickly to a change to a more suitable diet and the faecal fat is normal or only slightly raised.

(3) *Tuberculous Peritonitis* This is easily confused with coeliac disease since both conditions present marked abdominal distension loose stools etc. The author has seen coeliac disease in a child whose abdomen had been opened a year previously under the misapprehension that the case was one of tuberculous peritonitis. In distinguishing one from the other the Mantoux intradermal tuberculin test is of great value because at the age one meets with coeliac disease (i.e. under five years) a positive test strongly suggests an active lesion and would support the diagnosis of tuberculous peritonitis while

if the test is negative and performed up to a sufficient strength (10 mg) tuberculosis can be almost certainly excluded. Total fat output estimations are also of value here in differentiating the two conditions.

(4) *Rickets* This condition is often associated with a large abdomen and bowel irregularity but rickets may be distinguished from cœliac disease by x ray examination of the epiphyses the fact that it responds to vitamin D therapy and that the total fat output is normal or only very slightly increased. Cœliac rickets occurs when rickets complicates a typical case of the disease (see above).

**Prognosis** In the author's view the disease is one in which recovery may be complete though in severe cases some tendency to fat intolerance may persist for a long time. As has already been mentioned some stunting of growth may occur in severe cases but the mentality should be normal though speech development is often delayed for a considerable time.

### Treatment

(1) *Diet* This constitutes practically the whole of the treatment drugs playing a relatively unimportant part.

In planning the diet for a case of cœliac disease two points must be observed —

(a) *Fat* must as far as possible be excluded.

(b) *Starch* must be given in a more digestible form e.g. dextrins and maltose in order to reduce fermentative changes in the bowel and so lessen the frothiness of the stools and the degree of abdominal distension. It will be clear therefore that the basis of the diet must be protein and carbohydrate in the form of dextrin maltose. This is just the opposite of what the child has usually been receiving as in the belief that it was rickety or possibly tuberculous fats in the form of cod liver oil cream eggs butter etc. have usually been pressed upon the child. The necessity of giving fat soluble vitamins is mentioned below.

### Specimen Diet

BEFORE BREAKFAST	Orange juice or grape juice sweetened with Dextrin Maltose
BREAKFAST	Skimmed milk with the addition of gelatine or powdered sodium caseinate (Protocol 1 drachm to each 3 oz. milk)

BREAKFAST— <i>contd</i>	Force or Grape-nuts or Kellogg's Cornflakes crisp toast or rusks or Mellin's biscuit with honey <i>no butter</i>
MID DAY MEAL	Raw meat juice 3-6 oz (alone or sweetened with Dextrin Maltose)
DINNER	Pounded chicken or rabbit or fish or under done scraped steak Custard or jelly or over ripe banana Water to drink <i>No potato or vegetable</i>
TEA	Skimmed milk with the addition of gelatine or sodium caseinate <i>e.g.</i> Protosol Rusk or crisp toast and honey or sponge cake <i>No butter</i>

*Skimming* should be carried out for several hours. There is no necessity to take steps to make the curd any more digestible as there is usually no difficulty in the digestion of protein. Though skimming the milk in this manner does not remove every particle of fat it is usually sufficient but if a more completely skimmed milk is considered desirable a skimmed dried milk or buttermilk (which is virtually a lactic acid skimmed milk) may be used instead. The custard should be made with skimmed milk. It will contain a very small amount of egg fat but this is usually tolerated.

*Gelatine* is a form of protein which is useful in the diet. It may be given in the form of calves' foot jelly or ordinary jelly or gelatine strips melted down and added to the milk. While it does not supply all the essential amino acids it is a valuable source of protein.

*Meat* This has long been recognised as the *pièce de résistance* of the diet and it is interesting to recall that during the war when meat was rationed coeliac disease was one of those diseases on the list for extra allowance. Raw meat juice is also most valuable not only for its nutritious qualities but for its value in combating anaemia.

*Starch* As already mentioned starch is badly tolerated in the form of bread porridge potato milk puddings etc. and these should be excluded. In dextrinised form *e.g.* crisp toast, rusks etc. it is well tolerated. Later as the stools improve potato crisps made without fat may be added.

*Vegetables* These should be excluded at least during the earlier part of treatment. Later they may be carefully added in a sieved form. They do not seem to be an indispensable article of food and the author has had a case of coeliac disease for over two years on a vegetable free diet who remained otherwise perfectly healthy. Probably this is because the vitamins mineral salts and hæmoglobin forming chlorophyll can be supplied in other forms.

*Fruit* This is best avoided except as fruit juice or ripe banana. The latter is a very valuable addition to the diet because it is rich in protein is fat free and when ripe and of an amber colour, most of the starch is in the form of Dextrin Maltose. The Americans

incorporate this food very largely in their diets for the treatment of cases of the disease

**How Long Should Diet be Maintained?** This depends on individual cases and the severity of the case when it first comes under treatment. As a rule treatment takes from several months to one year, and very severe cases will need longer. No fear need be entertained in such cases as children can thrive, put on weight and remain perfectly healthy on a diet completely devoid of fat, provided that protein and carbohydrate are supplied in adequate amounts and the fat soluble vitamins included. When the child commences to gain in height and weight, and the stools have improved small quantities of fat may be added carefully the additions being controlled from time to time by examination of the fecal fat much in the same way as the diabetic diet is controlled by examinations of the urinary sugar. Following such increases of fat an attempt may be made to add well cooked cereals rice arrowroot, etc. Finally, a gradual return to normal diet may be made though usually a close watch has to be kept on the amounts of fat and starch that are given to the child for a number of years afterwards.

(2) **Vitamins** It will be clear from the diet outlined above that the addition of vitamins to the diet is imperative. The author has seen cases of coeliac disease suffering from rickets tetany and scurvy, all of which are manifestations of vitamin deficiency.

(a) *The fat soluble vitamins A and D* are best given in the form of one of the vitamin concentrate preparations e.g., halibut liver oil radiostoleum adexolin etc. The small amount of fat in these is very digestible and does not seem to upset the child. On the whole, therefore they are more satisfactory than preparations of irradiated ergosterol e.g., ostelin, calciferol, etc., since these do not contain vitamin A.

(b) *Vitamin C* is added, as mentioned above, in the form of orange juice or grape juice.

(c) *Vitamin B* does not seem to be so important and is not so restricted in the diet as the other vitamins, but it may be added possibly with advantage in the form of a small amount of Marmite.

(3) **Drugs** These, on the whole, play a minor part in treatment.

(a) *Iron* A dietetic shortage usually does not occur when

raw meat juice is given as advised above. If a secondary anemia develops as occasionally occurs in spite of this a salt of iron (e.g. ferri et ammon. cit. gr 3-5 aq ad 5i) may be given t.i.d. in the milk.

(b) *Opium*. Though this is sometimes recommended the author has never seen any advantages from it. The slight increased frequency of the stools in celiac disease is due to the malabsorption of fat to a large extent and since instead of excessive peristalsis there appears to be rather a paralysis of the gut it seems irrational to employ opium. It may also have the undesirable effect of diminishing the appetite which is always a difficult problem in celiac disease.

(c) *Pancreatic Extracts*. The administration of these has been almost universally abandoned since as already mentioned above the digestion of the food is not at fault but merely the absorption.

(d) *Bile Salts*. Since these not only play a part in the digestion of fat but also in its absorption the administration of these is more rational than the giving of pancreatic extracts. It has been shown however that there is no lack of bile in the intestine and the author has not been impressed with the results obtained by the administration of bile salts.

(e) *Calcium*. Pritchard advocates the administration of large doses of calcium by mouth in the form of prepared chalk so as to counterbalance the loss of base by the bowel. At the same time he allows larger amounts of fat than is usually advocated stating that the additional calcium will combine with the excess forming soap curds and making the stool firmer. The present author has no experience of this method.

(4) *Nursing*. In celiac disease as in pneumonia the survival of the patient is often more a triumph for the nursing staff than for the physician. The triumph is the more noteworthy in that whereas pneumonia is a disease of days or weeks celiac disease is one of months or years and the nursing of a single case is a vocation in itself. As a rule at the beginning of treatment the children do better in institutions rather than in their own homes since the child seems more easily able to acclimatise itself to the discipline and ordered life which it must follow if success is to be achieved.

Finally though the road may seem a long one no one can deny that the goal is worth attaining that of an apparently incurable and crippled child restored to normal health. In the

words of Leonard Parsons, " the disease and its treatment have a peculiar fascination, because by dogged perseverance and refusal to admit defeat, an apparently hopeless invalid can be transformed into a useful member of child society, able, on reaching adult years, to take his or her allotted place in the world "

## CHAPTER XV

W R I COLLIS

### CERTAIN ERRORS OF METABOLISM ASSOCIATED WITH HEPATOMEGALY

(Von Gierke's Disease Gaucher's Disease—Niemann Pick Disease)

#### Von Gierke's Disease

THIS condition has also been called hepatomegalia glycogenica and nephrohepatomegalia glycogenica at present it seems simpler to call it after Von Gierke who first described it in 1929

The disorder is characterised by enormous enlargement of the liver which commences during the first year of life The condition sometimes occurs in more than one child of the same family

The liver becomes gradually greatly enlarged it is hard and smooth and the edge is clearly felt The spleen is not enlarged but the left lobe of the liver may be mistaken for it as the latter fills the costo phrenic angle and passes under the left costal margin and down to or even below the umbilicus In some of the reported cases the kidneys have also been enlarged and palpable

The cause of the condition appears to be a disorder of carbohydrate metabolism Due to the lack of the glycogen splitting ferment the liver cannot dispose of stored glycogen which hence gradually accumulates so that eventually the liver is filled with masses of the carbohydrate Glycogen is also sometimes found in these cases in excess in the kidneys heart and brain In consequence of this state of affairs the child tends to be constantly on the verge of hypoglycæmia The urine may contain ketone bodies and the blood sugar be low and unaffected by the injection of adrenalin If a blood sugar curve is done after administration of a glucose meal a high prolonged hyperglycæmic curve is obtained due to failure on the part of the already overfilled liver cells to accommodate further glycogen

**Prognosis.** The condition is not incompatible with growth and development and several of the cases described in the literature are still alive. There is a tendency for these children to be more susceptible to intercurrent infection than normals, and if kept in hospital careful isolation from possible infection is necessary.

**Diagnosis.** Diagnosis rests on the finding of the enlarged liver, without an enlarged spleen, ketonuria which is unaffected by addition of carbohydrate in the diet, a low blood sugar, a high blood sugar curve following the administration of glucose, and finally the finding of liver cells full of glycogen by liver puncture.

**Treatment.** So far no specific line of therapy—such as the administration of adrenalin—has proved effective

### Gaucher's Disease and Niemann-Pick Disease

These conditions are both classified as disease due to errors of lipid metabolism. Their ætiology is obscure, but it is generally considered that they are due to the presence of certain fatty products which have failed to be broken down in the usual way and their accumulation in the tissues.

**Gaucher's Disease** occurs in different races, is probably familial and is more common in females than males. Usually it appears in children over two years of age, is slowly progressive till death occurs during the second decade.

The disease is characterised by enormous enlargement of the spleen which may appear to fill almost the whole abdomen. The liver is also much enlarged. The skin is characteristic, brown or yellow areas appearing on the face and neck and brown patches on the nasal side of the conjunctivæ. As the disease progresses hæmorrhages tend to occur from the mucous membranes and under the skin. Sometimes the bones become infiltrated and fragile, and fracture. An important feature is the leucopænia which always accompanies the disorder. The accumulation of the lipid in the reticulo-endothelial cells of the liver and spleen is the most characteristic feature of the disease. The cells (the Gaucher cells), when stained, present a specific appearance—appearing grouped in pyramids of pale yellow colour.

There is no treatment for the disease which is progressive and fatal.

Niemann Pick Disease occurs only in Jews is familial affects girls more often than boys and commences with the first few months of life

As in Gaucher's disease there is great enlargement of the spleen and some enlargement of the liver. The disease is characterised by the presence of large foam cells which are distended with lipoid and have specific staining reactions to Sudan III and Nile blue. They are found in the liver spleen lymph nodes brain etc. The skin becomes discoloured. Leucocytosis is present. Sometimes the condition is associated with amaurotic family idiocy. There is no specific treatment.

The diagnosis rests on the Jewish parentage of the child the age of the baby the splenic and liver enlargement the leucocytosis and finally upon splenic puncture. The latter procedure is the final arbiter in doubtful cases as when the splenic pulp so obtained is stained the *foam cell* will appear different to the Gaucher cell. Before performing splenic puncture a Wassermann reaction should always be done as enlargement of the liver and spleen due to syphilis is more common than any of these rare diseases.

## SECTION IV

### CHAPTER XVI

C. J. McSWFENEY

#### COMMUNICABLE DISEASES OF THE FIRST YEAR OF LIFE

(Whooping Cough—Measles Prevention of Measles Serum Prophylaxis, Serum Attenuation Placental Extracts—Diphtheria Nasal Laryngeal Faucial Serum General Treatment Prevention Control in Children's Wards, Table of Differential Diagnosis from Follicular Tonsillitis Quinsy Vincent's Angina—Scarlet Fever Prevention Control in Wards—Cerebrospinal Fever Pathology Signs and Symptoms Table of Differential Diagnosis from Pyogenic Meningitis Tuberculous Meningitis Anterior Poliomyelitis Epidemic Encephalitis—Small Pox and Vaccination Post vaccinal Encephalitis—Erysipelas Neonatorum Other Situations)

THE infant at birth and for some six months after enjoys a peculiar freedom from attack by the causative agents of diphtheria scarlet fever mumps and measles. This is said to be due to circulating antibodies of maternal origin which like all protective substances passively acquired disappear completely with the lapse of time. It is not understood why this congenital immunity does not apply to whooping cough small pox chicken pox and erysipelas.

#### WHOOPIING COUGH (PERTUSSIS)

During the first six months of life whooping cough is the infectious disease most commonly met with and at this age it is a very serious condition. Although the spasms of coughing may produce mechanical effects *e.g.* umbilical and inguinal hernia, rectal prolapse severe epistaxis subconjunctival hæmorrhage, etc. it is the respiratory, nervous and alimentary complications of this disease which kill. Laryngitis severe enough to merit operative interference may usher in whooping cough but this fortunately is not common. Broncho pneumonia developing usually during the paroxysmal stage makes pertussis one of the most fatal diseases of infants, the case mortality from this complication being sometimes as high

as 40 per cent. Survivors are left with lung legacies *e.g.* chronic interstitial pneumonia which may prove the starting point of pulmonary tuberculosis bronchiectasis or chronic bronchitis and emphysema in later life. Convulsions are quite common during a whooping cough broncho pneumonia but may occur in the absence of this complication. It is not known whether convulsions in whooping cough are toxic or asphyxial in origin (some authorities think they are due to tetany) but the important point is that three out of every four children who develop them die. An infant with whooping cough may manifest severe signs of gastro-enteritis. Children so affected rarely recover.

There can be little doubt that pertussis is caused by the Bordet Gengou bacillus. The incubation period is less than a week as a rule and the disease is most infectious in the early catarrhal stage when diagnosis is most difficult. A persistent hacking cough in an infant with disproportionately slight physical signs in the chest should always arouse suspicion of incipient pertussis. If the cough tends to become spasmodic during the next day or two if the child's face becomes congested during the spasms and if he vomits after a bout of coughing the disease is almost certainly pertussis. In this stage isolation is vitally necessary for infectivity is at its maximum. The diagnosis can be rapidly confirmed by the use of cough plates. A Petrie dish with a special medium containing human blood is held at a distance of 4 or 5 inches from the child's mouth during a paroxysm and immediately afterwards despatched to the laboratory. If the cough proves ineffectual it is permissible to re-expose the same plate on successive occasions within a few hours of the first attempt but delay in despatch of the inoculated plates should be avoided. A positive diagnosis may be possible in twenty four forty eight or seventy two hours by this means. A negative result is no criterion of freedom from pertussis any more than a negative throat swab excludes diphtheria.

When the characteristic whoop makes its appearance the diagnosis of pertussis is of course simple but as this may not be for several days or even for the whole course of the attack the use of cough plates in the early stages is of considerable diagnostic assistance. Their use in children's hospitals where recognition of pertussis at the earliest possible moment is of vital importance should be routine. In determining release

from isolation after an attack cough plates are also of service

**Treatment** The treatment of whooping cough involves a good deal of care and management. No drug has yet been discovered which shortens its course. Rest in bed and strict isolation in a well ventilated and sunny room is desirable. An adequate supply of fresh air with freedom from draughts should be maintained, the temperature of the room being from 60°-65° F. The patient should wear a flannel nightgown with a light chest jacket of Gamgee tissue. The old fashioned remedy of rubbing the chest with camphorated oil is to be recommended. The child should be supported during a paroxysm, and a receptacle held ready for the vomit which follows it. A small feed should be given about a quarter of an hour after a paroxysm. Regular feeding in pertussis is impossible and, as the vomiting can speedily bring about a severe degree of malnutrition, the milk feeds must be given as frequently as the paroxysms allow large feeds being avoided. Five per cent glucose orangeade may be given liberally. If gastro enteritis supervenes the milk should be diluted or peptonised and albumen water or whey may have to be substituted temporarily for the diluted or peptonised milk in severe cases. If the infant continues to vomit glucose salines (5 per cent) should be given by the rectum and the stomach washed out with a weak solution of bicarbonate of soda.

A sedative cough mixture containing a minum of Tr. Bella donna and 2 or 3 of Tr. Camph. Co. and Tr. Ipecac. with some syrup, should be given three times a day. For severe spasms a grain each of the three bromides and chloral hydrate can be given quite safely every four hours. Sometimes a small dose of luminal (gr.  $\frac{1}{2}$  twice or three times a day) may be given safely to a child one year old) acts like a charm but routine use of this toxic drug is to be deprecated.

Treatment by vaccines is only of value in the early stage of pertussis. I have had excellent results with dissolved vaccines and have not seen any reactions follow their use. After exposure to infection larger doses of the vaccine have either protected or been followed by very attenuated attacks.

An intradermal test of susceptibility to H. pertussis has been described recently by Patterson and Bailey, the antigen being 0.1 c.c. of a special vaccine prepared by Sauer. This test has been investigated in the wards of Cork Street Hospital by Dr

O'Brien, who found that it was a reliable, though not infallible, index of susceptibility. Sauer has described a technique of active immunisation against pertussis consisting of eight injections of his vaccine at intervals of a few days, but his work still awaits confirmation by field trials on a large scale.

### MEASLES

This disease is, next to whooping cough, the commonest of the infectious fevers met with during the first twelve months of life. The causative agent is a filterable virus which is coughed and sneezed into the air at a distance of several feet from the infectious patient. A carrier state does not exist in measles. Infection is contracted by the inhalation of infected droplets which have been sprayed from the nose and throat of a case of measles, or more rarely carried on the hands or gown of a nurse who has failed to observe an aseptic technique when attending on a measles patient. The infectivity of measles is greatest in the pre-eruptive or catarrhal stage, and it diminishes rapidly after the appearance of the rash. It is almost certain that in the post-eruptive broncho-pneumonia of measles the patient cannot transmit measles. The uncomplicated case of measles is probably not infective a week after the appearance of the rash.

The clinical features of measles in infants are too well known to merit special description here. The importance of associating catarrh of the respiratory passages accompanied by lachrimation and photophobia with incipient measles cannot be over-emphasised. When an infant shows signs of respiratory catarrh the practitioner should never omit to examine the buccal mucous membrane. In measles it is uniformly red, and the characteristic Koplik's spots are present at least twenty-four and sometimes forty-eight hours before the rash of measles appears.

Measles is often ushered in with marked laryngeal symptoms, and here again the reddening of the buccal mucosa and the detection of Koplik's spots will assist in diagnosis. The temperature in the pre-eruptive stage of measles, as a rule, steadily declines for three to four days from onset, but as soon as the rash begins to appear it rises to a level not before attained ( $103^{\circ}$ – $104^{\circ}$ ) and remains high while the rash is appearing over the body. Delay in the appearance of the rash is not

uncommon in young infants. A hot bath—to which a little mustard has been added—given in front of the fire together with the administration of small doses of brandy is often effective in stimulating a reluctant rash to come out.

Failure of lysis to appear with fading of the rash nearly always means the onset of a complication, most frequently that of broncho pneumonia. Physical signs in the chest are generally well marked and the temperature, pulse and respirations usually remain at a very high level during the course of the broncho pneumonia. This state of things may continue unabated for weeks, but favourable cases terminate in ten to twelve days. These cases of broncho pneumonia following measles demand skilful and patient nursing and should when ever possible be treated in hospital, preferably in a cubicle. In private practice the mortality is exceedingly high.

Another very serious complication is enteritis, which has its origin in the intestinal catarrh so often a symptom of measles. If the stools contain much mucus or if they are bloodstained, faecal swabs should be examined for organisms of the dysenteric group.

Otitis media is a very common complication in measles affecting infants. Eye complications are quite common in neglected children. Purulent conjunctivitis with much œdema of the lids and profuse discharge may persist for a long time and keratitis, corneal ulceration and permanent defective vision may follow. These eye discharges are infectious and unless an aseptic nursing technique is rigidly observed will spread to other patients in the ward.

**Treatment.** Measles should be treated in a well ventilated airy room free from draughts, heated by a coal fire and kept at a temperature of 60°–65° F. The room should contain a minimum of furniture and the cot should be accessible at all sides. The clothing and bed clothes should be light, one or two blankets provide sufficient covering with a hot water bottle. Daily bed baths are essential and whenever the temperature exceeds 103° F. the patient should be tepid sponged. The toilet of the mouth demands special attention which must be very gently given because of the inflamed state of the buccal mucosa. Gentle swabbing with cotton wool soaked in a 1 per cent. bicarbonate solution is all that should be attempted. Nasal douching does more harm than good. Septic and ulcerative conditions of the mouth and nostrils are

prevented by smearing the lips and nasal orifice with a little pure vaseline. Discharges from the nose, mouth and eyes should be received on cotton wool swabs and burnt. Irrigation of the conjunctival sacs is unnecessary unless severe conjunctivitis is present when they should be washed out with boracic lotion or normal saline at least four hourly. The eyelids should be smeared with a little mild emollient *e.g.* pure vaseline in these cases. If the eye discharge persists 1/10 000 hydrarg. perchlor. should be used as an irrigant.

The diet in measles is very important. Enteritis is an exceedingly serious complication in infants and is much easier to prevent than to cure. Milk should always be given diluted to infants in the acute stage of measles. The stools should be carefully watched for curds, the appearance of which should be taken as an indication for peptonisation or citration. Glucose orangeade or lemonade should be given freely between the regular feeds. The treatment of enteritis following measles does not differ from that recommended for enteritis with pertussis (see p. 149).

It is a safe rule to keep the child in bed for a week after the temperature has settled and indoors for a further few days.

In convalescence cod liver oil should be ordered. Proprietary vitamin preparations are often stated to be of special use in warding off complications in measles but there is no evidence that this is so. Amidopyrine has been used in the treatment of measles and is claimed by some to be specific in its action on the measles virus. It is given in gr.  $\frac{1}{4}$ -1 doses to infants up to one year of age. It is said to cut short the pyrexial stage but it does not lessen the risk of complications. It may cause agranulocytosis. The use of this toxic preparation is to be deprecated.

If laryngitis occurs at the onset of measles the infant should promptly be given at least 20 000 units of diphtheria antitoxin. A concurrent laryngeal diphtheria is by no means uncommon in measles. Serum administration should never be withheld pending the result of a bacteriological examination. Steam is helpful in the early cases and so is the mixture referred to in connection with the treatment of laryngeal diphtheria (p. 157). In hospital practice direct laryngoscopic examination enables a laryngeal culture to be taken and any membrane or mucus seen in the glottis can be aspirated at the same time. If the laryngeal symptoms persist in spite of suction, intubation or

tracheotomy may be necessary. The mortality for cases requiring operative treatment is very high.

Broncho pneumonia is best treated in an oxygen tent. Nasal oxygen is of service should an oxygen tent be not available. Failing this, an adequate supply of fresh air is the most potent therapeutic agent. The windows must be open at the top and the cot should be so placed that a current of fresh air is always passing just over the child's head. Children nursed in 'fuggy' over heated rooms invariably die. A light jacket of Gamgee tissue or antiphlogistin should be applied to the chest and the child should be nursed partially propped up. Strychnine, camphor and brandy are the only drugs of service for routine use, and should be prescribed in doses of gr  $\frac{1}{120}$  gr 1 and ℥  $\frac{1}{2}$ –℥  $\frac{1}{4}$ , respectively, every four hours. Coramine (17 cc) is a useful emergency stimulant. Occasionally quite spectacular results follow the administration of 10 000 units of polyvalent anti pneumococcus serum. Two good nurses (one for the day and one for the night) are essential if the case must be treated at home. The nurse should be warned against permitting relatives to remain in the sick room.

### Prevention of Measles

The various methods by which measles can be prevented or attenuated have already been referred to.

When a hospital ward becomes infected, complete protection of all contacts is the aim. Apart from hospital practice, attenuation of the attack after exposure to infection is the method of election.

**Serum Prevention.** For the purpose of protecting a child exposed to measles from developing an attack the serum (1) of patients convalescent from measles collected preferably one week after the temperature has settled, or (2) of healthy young adults (e.g., nurses or students) who have had measles previously, may be used.

These immune sera contain antibodies which when introduced into the system within four days of exposure to measles, confer complete protection, provided the serum used is a potent one. Convalescent serum is naturally richer in antibodies than the serum of a person whose attack of measles occurred some years previously.

For an infant of twelve months or under, whom it is desired to protect against measles, the dose of convalescent serum

should be in the region of 5 c c. If adult serum is used the dose ought to be 10 c c. If neither is available the injection of 20 c c. of the citrated whole blood of one or other parent who has had measles should be tried. 1 c c. of a 10 per cent. sodium citrate solution should be added to every 10 c c. of blood taken from the parent.

Serum or citrated whole blood should be administered intramuscularly within four days of exposure to infection. It is important to remember that serum protection merely confers a passive immunity of not more than three weeks' duration.

**Serum Attenuation** If it is desired that the child should be permanently immunised to measles this is the method of choice. The procedure is the same but the inoculation is deliberately withheld until the fifth or sixth day after exposure so that the immunity conferred will be only partial allowing a modified attack of measles to occur. This attack however is sufficient to produce a permanent immunity to measles. Where serum is scanty attenuation may also be produced by giving during the first four days following exposure a smaller dose than that necessary for complete protection.

In calculating the period which has elapsed since exposure it is wise to assume that the infecting case was active at least four days before his rash appeared.

**Placental Extracts in Measles Prophylaxis** In America, McKhann and Chu and others have recently reported the use of an immune globulin derived from human placentas for producing a passive immunity to measles. Confirmation of these results was reported in April 1936 by Dr Joe of London who says that the reagent is at least equal to adult serum.

I have found the immune globulin preparations of the Lederle laboratories quite as efficacious as measles serum in the prophylaxis and attenuation of measles.

## DIPHTHERIA

Infection with diphtheria bacilli under the age of one year is apt to take either the nasal or laryngeal form faucial diphtheria being much rarer.

### Nasal Diphtheria

The clinical evidences of this condition consist in a discharge from the nose often blood stained and accompanied by some

excoriation of the nostrils. If the infection be confined to the nose, the constitutional symptoms are trifling, and, once the child has been segregated, given a small dose of antitoxin (10,000 units), its arms splinted so as to prevent aggravation of the nasal condition by picking, little else requires to be done. These cases are really more properly described as diphtheritic rhinitis, and the risk of complications arising is negligible. Their importance from the epidemiological standpoint, however, is considerable. Introduced into, or occurring unrecognised in, a children's ward, such cases are capable of causing an outbreak of diphtheria which, in other infants may take the more severe laryngeal form, or, in older children affect the fauces and nasopharynx.

It is comparatively common for swabs taken from the running noses of infants to be reported "positive for bacilli morphologically resembling the Klebs Loeffler organism." Such a finding justifies neither a diagnosis of nasal diphtheria, nor the removal of the child to a diphtheria ward. In many of these cases the organism found will not kill a guinea pig. The organism is avirulent and is incapable of causing symptoms of diphtheria in the patient or anybody else.

The following procedure is advised in dealing with cases where a nasal discharge, reported positive for Klebs Loeffler bacilli, represents the sole evidence on which a suspicion of diphtheria is founded. Perform a Schick test and after allowing the toxin to be fixed in the cutaneous tissues (*i.e.*, about twelve hours later) give a small dose of diphtheria anti-toxin (say 10,000 units). Segregate the child or if impossible, nurse it on the bed isolation principle. Submit the culture obtained from the nose for virulence testing—if it be avirulent the child cannot be suffering from clinical diphtheria and bed isolation can be discontinued, though he should be immunised if Schick positive. If it be virulent and the Schick test positive, the child must be regarded as a true case of diphtheria and should be removed to an isolation cubicle or fever hospital and treated accordingly, further antitoxin will be unnecessary unless faucial or laryngeal symptoms arise.

If it be virulent and the Schick test negative, the child is a nasal carrier, and should be removed from the ward and kept isolated until he ceases to carry the germ.

If, in spite of the initial dose of antitoxin, any of the following symptoms arise—croupiness, hoarseness, aphonia, exudation

on the tonsils faucial œdema (especially if the last two be associated with cervical adenitis)—a further dose never less than 30 000 units of antitoxin should immediately be administered irrespective of the age of the child

### Laryngeal Diphtheria

This is the most fatal form of diphtheria to very young children a fact attributable to the relatively small size of the glottis at this age and the comparative ease with which it may be occluded by membranous or inflammatory swelling. This form of diphtheria does not kill by toxæmia the patient may die before operation from asphyxia during operation from shock after operation from broncho pneumonia. Diphtheria of the larynx begins with fever hoarseness and a spasmodic dry and barking cough with some dyspnoea. These symptoms are sometimes due to catarrhal laryngitis but if diphtheritic in origin the spasms of coughing speedily increase in severity and the intervals between them diminish. When a spasm is not in progress the breathing is noisy or stridulous and the respiratory rate is increased. There is recession of the intercostal spaces supraclavicular and epigastric regions the lips of the child become progressively cyanosed and he becomes increasingly restless. This stage of laryngeal diphtheria may last from a few hours to a few days and if unrelieved the child passes into the final stage when the respirations become still more frequent but shallower stridor becomes inaudible recession diminishing in proportion to the degree of asphyxiation of the child who is now too exhausted to struggle.

Recognition of these cases at an early stage is literally vital as all forms of operative interference are attended by a high mortality in infants. It is not possible clinically to distinguish catarrhal laryngitis from the early stage of laryngeal diphtheria if direct laryngoscopy is not practicable all these cases should receive a precautionary dose of antitoxin. Laryngismus stridulus is easily recognised by its intermittent character and the fact that the child is perfectly well between the spasms. Retropharyngeal abscess is detected by palpating the posterior pharyngeal wall and broncho pneumonia by examining the chest. The differential diagnosis of laryngeal diphtheria from the catarrhal stage of measles is discussed under measles (p. 150).

An infant suspected of developing laryngeal diphtheria should be given at least 20 000 (preferably 30 000) units of antitoxin and put into a steam tent at once. To an infant of nine months an anti spasmodic mixture containing Tr. Bella donna ℥ i Tr. Ipecac ℥ ʒ Tr. Camph. Co. ℥ x and Pot. Iodide gr. iii well sweetened with syrup can be given four hourly. A warm but light application to the throat helps to loosen the membrane. In favourable cases (which include all cases recognised early) the membrane ceases to spread, loosens and as it is coughed up in shreds all symptoms of respiratory embarrassment disappear. Aspiration of the loosening membrane through a laryngoscope is helpful in some cases and if it does not relieve the condition direct intubation can be performed at the same time. In intubation a vulcanite tube introduced into the larynx maintains an airway for several days until the detached membrane is ready to be coughed out. The coughing-out of the membrane often coincides with the expulsion or extraction of the tube. Intubation is essentially a hospital procedure. If the instruments for suction and intubation are not available or if intubation fails to relieve the condition (as occurs when the obstruction extends low down into the trachea or even the bronchi) tracheotomy must be performed.

### Faucial Diphtheria

Diphtheria may attack the fauces alone or may occur with nasal and laryngeal lesions. It is uncommon for infants under one year to suffer from faucial diphtheria alone. The disease begins as a follicular tonsillitis, small discrete spots of exudate spreading rapidly in circumferential fashion to become scattered patches of membrane which later coalesce to form a continuous investment of the tonsils, faucial pillars, adjacent soft palate and uvula. Unchecked by early serum administration the diphtheritic process can spread from the faucial ring to the hard palate anteriorly and the nasopharynx posteriorly in the space of forty-eight hours. Involvement of the nasopharynx is rapidly followed by the appearance of nasal discharge which is often blood stained. Faucial and nasal diphtheria is associated with more toxæmia than any other variety. With the gravis type of infection so common in Dublin in recent years some degree of faucial œdema is the rule. The diphtheritic membrane has no characteristic colour,

but it sometimes has the pearly grey hue described in text books—oftener it is greenish black. If any smell be detectable from a diphtheritic membrane it is of a putrescent character. When detached such a membrane leaves a bleeding surface a point of distinction from tonsillitis. There is never any suggestion of ulceration or loss of tissue (as in Vincent's angina) in the appearance of a diphtheritic throat. Inflammation of the surrounding tissues is rare in diphtheria and neither pain nor pyrexia are marked features of the disease—points of contrast to the streptococcal pharyngitis which is often confused with it. In quinsy the presence of stringy mucus over the swollen tonsil often gives rise to a suspicion of diphtheria but the asymmetrical bulging with much surrounding inflammation pain and pyrexia generally enables the correct diagnosis to be made (see Table V).

Cervical adenitis is never long delayed in faucial diphtheria, it is toxic in origin the degree of gland enlargement being proportional to the amount of toxin absorbed. In cases of four days standing or more the glands form a collar around the neck. Sometimes parents have mistaken the condition for mumps. In the toxic adenitis of diphtheria the glands are not unduly hard the bull neck seen in the severest forms of this disease consisting of a puffy swelling of the glands and cervical tissues with no induration. Delayed diagnosis in these toxic cases usually involves a fatal termination.

Toxic adenitis in diphtheria does not suppurate unless a superadded septic infection is present. The glands subside quickly as antitoxin is absorbed into the circulation. If exudation is present on the tonsils of a young child who is not suffering from scarlet fever the disease is almost certainly diphtheria. If the slightest speck is present on the tonsil with croupiness the same diagnosis applies. Unilateral tonsillar patching in a child is almost always diphtheritic. Any concomitant enlargement of the cervical glands or the presence of nasal discharge strengthens the suspicion that exudate on the throat is diphtheritic. Absence of pain moderate pyrexia pallor and the presence of albumen in the urine are further corroboratory signs.

Two golden rules in connection with sick children are —

- (1) Never omit to examine the throat of an ailing child and
- (2) Never take a swab from the throat until after antitoxin has been given

Table V—Differential Diagnosis of Faucal Diphtheria

	Faucal Diphtheria	Follicular Tonsillitis	Quincy	Vincent's Angina
Nature	Adherent spreading membrane	Purulent exudate not tending to spread	No deposit over swollen boggy tonsil. Possible stringy mucus	Superficial ulceration with slough
Colour	Often poorly grey may be white or black	Yellowish	Yellowish	Greenish
When detached	Bleeds	Does not bleed	Does not bleed	Does not bleed
Smell	Putrid	Nil	Nil	Generally nil may be foul
Site	May be unilateral, if bilateral spreads medially towards uvula	Generally bilateral	Unilateral	Generally bilateral
If untreated	Spreads to faucial pillars, palate, uvula and nasopharynx	Remains confined to tonsils	Remains unilateral with increasing swelling until rupture or incision	Does not change much in extent from day to day
Surrounding tissues	Pale oedematous in grave infections	Red and inflamed	Intensely swollen and oedematous	Not inflamed
Cervical glands	Enlarged in proportion to extent of membrane	May not be enlarged or only slightly so	Enlarged on affected side	Often unaffected
Associated conditions	Soft	Rheumatic pains	Hard	Often enlarged
Temperature	Often nasal discharge and/or laryngitis in infants	Markedly raised	None	Stomatitis and/or ulceration around teeth
Temporature	Only slightly raised if no laryngeal involvement	Markedly raised	Markedly raised	Slightly raised or normal
Colour of patient	Inert depressed	Restless complaining	Very fit and rosy	Often unaffected
Urin	Pale	Clouded	Plentiful	Often normal
Swallowing and mastication	Absent	Present	Considerable	May be absent
Organisms present in smear or swab	Unaffected	Painful	Very painful may be difficult	May be difficult but generally unaffected
	Diphtheria bacilli	Hemolytic streptococci	Uremolytic streptococci	Is fusiform and spirilla Vincent's

## Other Varieties of Diphtheria

Diphtheritic infection of the conjunctiva the skin or the external genitals though rare is occasionally encountered. Toxæmia in these cases is not marked and paralytic or cardiovascular complications are the exception. The possibility that *C. diphtheriæ* may be the causative organism should be kept in mind when a chronic inflammation especially if associated



FIG. 14.—Conjunctival diphtheria (Cork Street Fever Hospital, October 1937.)

with the formation of a membrane occurs in any of these situations. A membranous investment of the conjunctivæ in a child certainly necessitates the taking of a swab. Prompt treatment with diphtheria antitoxin clears up these conditions within a few days. Figs. 14 and 15 show a concurrent diphtheritic infection of the conjunctiva and the skin behind the ear in a case successfully treated in Cork Street Fever Hospital in October 1937. Swabs from the conjunctiva, skin behind ear and nose in this case yielded a pure culture of *C. diphtheriæ*. Within twenty-four hours of antitoxin adminis-

tration this case had so much altered that it was not considered worth showing to a class of students !

A sloughing membrane on the vulva particularly in a debilitated slum child may resist local treatment for weeks but if treated with 4 000 units of diphtheria antitoxin may disappear overnight In one case under my care a child less



FIG. 15.—Cutaneous diphtheria (from same patient as Fig. 14).  
(Cork Street Fever Hospital Dublin 193 )

than a year old had diphtheritic infection of the throat nose larynx conjunctiva skin and vulva but recovered

(For Figs 14 and 15 I am indebted to Mr J J Murphy of the Richmond Hospital and to my assistant Dr H R Rogers who suggested that the case should be photographed )

### Treatment of Diphtheria

**Serum** Administration of serum is the most important part of the treatment of diphtheria The aim should be to give one single adequate dose of antitoxin as early as possible Children so treated on the first day of their disease recover those not so treated die in proportion to the delay in administering serum It is literally fatal to withhold serum until bacteriological confirmation of the clinical findings is available The minimum therapeutic dose of antitoxin is 4 000 units the maximum

single dose which can be given conveniently by the intramuscular route is 60 000 units. The latter is reserved for severe faucial and nasal cases with much glandular enlargement and profound toxæmia. A moderate faucial case with membrane confined to the tonsils should receive at least 20 000 units. Cases of laryngeal diphtheria (who rarely exhibit toxæmic as distinct from asphyxial symptoms) require from 20-30 000 units in a single dose. Diphtheria confined to the nose is adequately dosed if 10 000 units is administered. Spread of membrane or increase of toxæmic symptoms indicate a second dose of antitoxin which should never be smaller than the first. A total dosage of 120 000 units may be taken as the maximum. It is doubtful whether larger amounts are of any value. For all cases other than the very toxic the intramuscular route is the best. The middle of the outer side of the thigh is the most suitable site for inoculation, a sharp sterile needle being plunged into the Vastus Externus muscle for about an inch. Successive injections may be given into alternate thighs. Where toxæmic symptoms are in evidence some of the serum may be given intravenously after the primary intramuscular inoculation. I have never seen a severe general reaction follow serum administration in an infant.

The general management of diphtheria involves keeping the child flat until all danger of cardiac and paralytic complications is past. This means the employment of some form of restrainer in the case of infants. Only one pillow should be allowed and the child should be spoon fed by a nurse. Enemas should be given every other day until the second pillow is granted which in mild cases will be at the end of three but in moderate or severe cases will be postponed for four five or six weeks. A third pillow is given a week after the second and after another week the child may be allowed up out of bed.

During the acute stage milk with plenty of glucose orangeade drinks is all that should be allowed. When the diphtheritic lesions have cleared porridge Benger's food milk puddings and mashed potatoes can be given and when the second pillow is granted the normal diet may be resumed. Bed bathing and the toilet of the mouth should be carried out with a minimum of disturbance to the patient who must be spared every effort. Local treatment of the throat is valueless upsets children and hence actually may be harmful.

### Prevention of Diphtheria

Susceptibility to diphtheria is determined by the Schick test which consists in the intradermal injection of 0.2 c.c. of a standardised diphtheria toxin. With infants it is rarely necessary to carry out a Schick test for after six months the great majority are known to be susceptible to the disease, although the maximum incidence is naturally amongst the older children who incur far greater risks of exposure to infection.

If it be desired to Schick test an infant as a preliminary to immunisation it is quite unnecessary to perform a control the test toxin merely being injected intradermally into the left forearm and the reaction read seventy-two hours later.

The optimum time to immunise children is at the age of nine months. The prophylactic used may be (1) toxoid antitoxin floccules (T A F) which is administered in three doses of 1 c.c. at weekly intervals, or (2) alum precipitated toxoid the dose of which varies from  $\frac{1}{2}$  to 1 c.c. given in two injections at intervals of at least a week. The so-called 'one shot method' is not to be trusted. T A F is a safe and reliable antigen which has never in my experience caused the slightest reaction in children. It is given subcutaneously, a suitable site being the skin over the insertion of the deltoid muscles. If for any reason it is desired to reduce the number of injections, alum precipitated toxoid may be given. This prophylactic is injected intramuscularly. Local induration is relatively common after alum injections and sometimes persists for a long time. Whatever prophylactic is used a Schick test (with control) should be performed about six months later. Should this post inoculation Schick test prove positive further injections should be given. If T A F has been used for the primary immunisation the posterior Schick test is almost always negative. Should it be positive only one further dose of T A F (1 c.c.) need be administered.

### The Control of Diphtheria in Children's Wards

When a case of diphtheria occurs in a children's ward the following procedure should be carried out —

(1) Remove the infectious case to an isolation cubicle where he should be nursed on strict barrier principles. If no cubicle is available removal to a fever hospital is necessary.

(2) Perform the Schick test on all remaining children in the ward  
 (3) Take swabs from the nose and throat of all children in the ward

(4) Swabs reported positive should be tested for virulence. When the Schick tests and the swab results are available it is possible to classify the ward contacts into four groups —

*I Schick + Swab +* These children are susceptible to diphtheria and are harbouring diphtheria bacilli. They should be given antitoxin at once and removed to isolation cubicles or a fever hospital.

*II Schick — Swab +* These children are insusceptible to diphtheria but are harbouring diphtheria bacilli. They should be isolated pending the result of a virulence test. If the bacilli prove virulent they should be retained in isolation (in a cubicle or fever hospital) until free of organisms. If the organisms are reported avirulent the child may be allowed to return to the ward.

*III Schick + Swab —* These children are susceptible to diphtheria but not infected. They should be kept in the ward and immunised with diphtheria prophylactic (T A F or A P T).

*IV Schick — Swab —* These children are insusceptible to diphtheria and not infected. They may be left in the ward without any treatment.

### SCARLET FEVER

The clinical features of scarlet fever seen in infants do not differ from those in older children except that the rash is generally less punctate in character and true desquamation does not occur in infants the skin merely showing a powdering rather than a peeling effect. Streptococcal complications are much more likely to occur in infants and of these the most fatal is broncho pneumonia.

Broncho pneumonia occurring in scarlet fever does not differ clinically from the same condition following measles. Otitis media suppurative adenitis septic skin conditions and in neglected cases cancrum oris are not uncommon complications of scarlet fever in infants.

The great majority of the fatal cases of septic scarlet fever which have occurred in Dublin in recent years were in infants and very young children.

**Treatment** In treating a case of scarlet fever the toilet of the mouth must be carefully attended to and if septic complications are to be avoided daily bed bathing is essential.

Streptococcal antitoxin (3 000–6 000 units corresponding to

10 or 20 c c) should be given intramuscularly at an early stage, in all except the mildest cases. This may be repeated if the temperature does not begin to decline within twenty four hours of administration. In severe cases more antitoxin may be given but it is doubtful whether a total dosage of more than 18 000 units confers any further benefit. Any exudation on the tonsils calls for the precautionary administration of diphtheria antitoxin as well as streptococcal antitoxin.

In septic cases tablets of prontosil album (or similar preparation) may be given crushed up in milk. Half a tablet three times a day may be prescribed safely for infants of a year old. More severe cases may receive 2 c c prontosil rubrum once or even twice a day but experience in this form of chemo therapy is tending to show that the non staining (album) preparations given by the mouth are therapeutically more efficacious. When prescribing prontosil sulphur derivatives (e.g. Epsom or Glauber's salts) should be forbidden as should any sulphur containing food (e.g. eggs). Rest in bed in a warm well ventilated room for at least three weeks is advisable.

During pyrexia only fluids should be allowed and it is well to supplement milk with glucose orangeade.

When the temperature has settled Benger's food milk puddings porridge vegetable soups and bread and milk can be given. It is probably better to withhold eggs until the danger of nephritis has passed.

Infants with scarlet fever should be nursed propped up and chills should be carefully guarded against. Otitis demands the instillation of sedative ear-drops but if the drum is seen to be inflamed an early paracentesis often saves much pain and limits middle ear mischief. Cervical adenitis in scarlet fever is best treated in the early stages with iodexunction. If suppuration seems inevitable an antiphlogistine poultice should be applied. It is not wise to be precipitate in opening suppurating glands when only a small quantity of pus has formed—a sloughing slowly healing wound is apt to follow premature incision in these cases of scarlet fever.

For the treatment of streptococcal broncho pneumonia see under Measles (p. 153).

### Prevention of Scarlet Fever

Susceptibility to scarlet fever may be determined by the Dick test the technique of which is similar to the Schick test.

except that a standardised toxic filtrate of the scarlatinal group of hæmolytic streptococci is substituted for diphtheria toxin. No control is necessary when testing infants. The test is read eighteen to twenty four hours after the intradermal injection of 0.2 c.c. of the toxin. Infants who are found to be Dick positive may be passively immunised by the administration of 2 000 units of scarlatinal antitoxin but this procedure is not recommended unless there has been definite exposure to infection.

Active immunisation against scarlet fever may be effected by a series of graded doses of scarlatinal toxin starting with 250 or 500 skin test doses and trebling each dose successively at weekly intervals until a total of 20 000 skin test doses is given. This dosage generally confers an immunity lasting at least two years when another Dick test may be done and a further course of inoculations given. Some authorities give up to 80 000 units in the first course of injections but for infants such high doses are not to be recommended. As the antigen employed is an unmodified toxin there is some risk of local and even general reactions. This tendency is considerably lessened if 0.2 c.c. of adrenalin be added to each immunising injection.

### The Control of Scarlet Fever in Children's Wards

When a case of scarlet fever occurs in a children's ward the patient should be moved to an isolation cubicle and all remaining children should be Dick tested. The Dick tests can be read in eighteen to twenty four hours and those found positive should receive not less than 2 000 units of scarlatinal antitoxin intramuscularly. This procedure is generally sufficient in preventing further cases arising but it is well to remember that the passive immunity conferred by the antitoxin wears off in three weeks after its administration. In the case of children who are likely to be in patients for long periods active immunisation against the disease (see above) should be commenced a week after the inoculation of scarlatinal antitoxin.

An attempt has been made recently to broaden our conception of scarlet fever. It has been alleged that the characteristic rash is only one manifestation—a toxigenic one—of infection with the hæmolytic streptococci of scarlet fever. Scarlatinal infection it is said may show itself as the classical punctate

exanthem in one child of a household as a tonsillitis without rash in another and as a fleeting erythema without tonsillitis in a third

A certain amount of support has been forthcoming for this view and already one meets in the journals such expressions as scarlatina without the rash. It is the opinion of the writer that this new hypothesis should be received with the utmost caution. It is by no means certain that a particular group of hæmolytic streptococci constitutes the sole ætiological factor in scarlet fever.

For many years scarlet fever has been recognised as a well defined clinical entity capable of causing a similar exanthem in exposed susceptibles. The appearance more recently of a benign form with a negligible mortality rate is not without precedent in the history of infectious disease. The community control of scarlet fever should it again become virulent would be impossible if the disease is allowed to lose its identity. It is true that hæmolytic streptococci may cause epidemics of sore throat in residential schools and institutions and that more serious sequelæ may be met with in these epidemics than with the prevailing type of scarlet fever but this is no reason for regarding all forms of streptococcal infection of the upper respiratory passages as aberrant types of scarlet fever. The task of identifying the various exanthemata as so many distinct clinical entities has been tardily accomplished and attempts to obscure the present clear cut conception of any one of them should not be lightly entertained.

## CEREBROSPINAL FEVER

### (Meningococcal Meningitis)

In this acute infection of the central nervous system the brunt of the infection is borne by the posterior portion of the base of the brain (*pons cerebellum and medulla*) and hence before the meningococcal nature of the disease was realised it was named the posterior basic meningitis of infants by Gee and Barlow.

The disease is often insidious in onset the symptoms very often dating from a fall on or other injury to the head. Meningococcal meningitis may occur in the early months of life but is more commonly met with in the second year than in the

first The younger the child the worse the prognosis The disease is apt to run a very chronic course with extreme wasting hydrocephalus central blindness extreme retraction of the head with exaggerated opisthotonus and spastic rigidity of the limbs Infants who survive often become imbeciles Cutaneous hæmorrhages are rare in cerebrospinal fever affecting infants The onset may be sudden but is usually spread over a few days with gradually increasing fretfulness screaming and possibly convulsions Gastro intestinal symptoms often occur and may be so severe as to divert attention from the central nervous system The temperature is usually high it tends to be irregular and its height bears no relation to the severity of the disease The child lies on his side crying out occasionally and resists being touched There is rigidity of the neck muscles and occasionally Kernig's and Brudzink's sign may be present Squint may occur rarely but ptosis is exceedingly uncommon in the acute stages

Acute cases may die rapidly within a few days of onset but the more common course is protracted Increasing rigidity of neck muscles and head retraction the assumption of the gun hammer position with considerable wasting supervenes and in this state the infant may linger for weeks The wasting is undoubtedly a trophic phenomenon Bullæ may appear on the limbs and trunk It is really amazing how long life may linger in the twisted emaciated bodies of the infants so afflicted

The diagnosis of cerebrospinal fever in infants cannot be made on clinical grounds in the early stages of the disease When a child is found to present signs and symptoms suggestive of acute meningitis a spinal puncture must be done as soon as possible No anæsthetic is required for this diagnostic puncture For general purposes the puncture is best made in the lumbar region in the intervertebral space above or below an imaginary line joining the crests of the iliac bones which passes through the 4th lumbar spine The puncture is most easily performed exactly in the middle line the needle being entered at right angles to the skin and pushed firmly through the ligament In infants it is wise to dispense with the stilette when introducing the needle especially at first puncture The canal is fairly superficial the ligament not tough and the spouting of the fluid when the meninges are pierced prevents the common mistake of pushing the needle too far in which of course always results in a blood stained fluid because of damage to

the venous plexus on the floor of the crani. A turbid fluid under pressure always indicates the administration of anti meningococcal serum. Light anæsthesia is advisable if it is desired to proceed to intrathecal therapy unless the child is comatose. The amount of serum given should be about 5 c.c. less than the volume of fluid withdrawn. It is most conveniently given by a syringe though the funnel (gravity) method is favoured by some. The important point is to give the serum slowly. The serum should always be warmed to body heat. The rate of inflow should be 1 drop per second. *To do this ten minutes are required to inject 70 c.c. a quarter of an hour to give 30 c.c.* In meningococcal meningitis the organisms are readily seen as Gram negative diplococci by direct examination but if scanty or absent a culture should be done unless other pathogenic organisms have been found by direct examination. Cells are markedly increased in meningococcal meningitis and are predominantly polymorphic. The sugar is always diminished or absent. Globulin is increased as it is in all forms of meningitis. The chloride content is not markedly lowered. A simple test for meningitis which can be performed at a bedside without any reagents is as follows. Put a finger over a test tube one third full of cerebrospinal fluid agitate the contents vigorously for half a minute and allow to stand. In a non meningitic fluid the supernatant froth subsides quickly but in meningitis (because of increased globulin) the froth remains and may persist for many hours. I have seen it present next day. The Nonne-Apelte test is also easily applied if a saturated solution of ammonium persulphate be available. Recently a ring test has been described which consists in layering some of the C.S.F. on to polyvalent anti meningococcal serum when a white ring appears at the junction of the two solutions.

**Treatment** When the diagnosis of meningococcal meningitis has been confirmed bacteriologically the plan of treatment should be to give an intrathecal and an intramuscular injection of serum every day for four days the fluid being examined each day for organisms cells and sugar. Diminution in the number of meningococci especially if accompanied by fragmentation and preponderance of intracellular meningococci and increase or reappearance of sugar are good portents. *Vice versa* increase in turbidity or in the amount of sediment of the fluid with continued absence of sugar and persistence of numerous extracellular diplococci are bad signs. If on the fourth injection

the fluid is clear and free of organisms serum therapy may be discontinued. If this is not so intrathecal therapy should be continued on alternate days for another three or four injections in the hope that the disease will be arrested. Intramuscular serum may be discontinued after the initial four days. If adhesions occur in consequence of repeated punctures in the lumbar region serum therapy can be given by the cisternal route. The operation is not entirely free of risk and novices should not attempt it without previous practice on the cadaver. A stout needle preferably graduated in centimetres is required. The patient should lie on his right side with the head flexed strongly on the chest. The puncture is made exactly in the middle line through the depression immediately above the axis vertebra. The needle is pushed upwards and slightly forwards in the plane formed by three points—the puncture the external auditory meatus (upper edge) and the glabella. A little less than 2 inches beneath the skin the occipito atlantoid ligament binding the posterior border of the foramen magnum to the atlas is met with and pierced. Immediately this happens the needle is in the cisterna magna. On withdrawal of the stylette fluid appears. If fluid does not appear it is better to withdraw the needle and start afresh rather than to poke about with the needle.

Increase of intracranial pressure contra indicates cisternal puncture because of the displacement downwards of the cerebellar vermis. Pushing the needle too far into the cistern may damage the floor of the fourth ventricle. According to F. C. Eve the distance at which fluid is struck in cisternal puncture can be gauged accurately by measuring the circumference of the patient's neck and dividing by nine.

If hydrocephalus occurs ventricular puncture (with or without serum therapy) may be done. The infant is laid on his side with the head projecting beyond the head of the table. An ordinary lumbar puncture needle is pushed through the skin just lateral to the angle of the fontanelle and having pierced the meninges is directed downwards in the plane of the coronal suture. The resistance to the passage of the needle suddenly gives when the needle enters the lateral ventricle and fluid—usually quite clear—comes freely. Amounts of 40–50 c.c. may be allowed to drain away. The operation causes no untoward symptoms and infants frequently finish a bottle feed while it is in progress. Alternate lateral ventricles may be drained on

consecutive days. The ventricle being drained should, of course, be uppermost during the operation. Should an infant need ventricular puncture, the chances of complete recovery are very remote.

Should a turbid C S F fail to show or to grow meningococci, other forms of pyogenic meningitis must be considered. The differentiation is entirely a matter for the bacteriologist. Pneumococcal or streptococcal infections elsewhere (e.g. in the lung, peritoneum, the ear, mastoid or sinuses) may give a clue. Influenzal meningitis, however, is rarely secondary to any clinically recognisable focus.

Tuberculous meningitis is often suspected on clinical grounds before the clear C S F with characteristic fibrin web, excess of lymphocytes, diminished chlorides and possibly tubercle bacilli clinch the diagnosis. Very gradual onset with increasing drowsiness, relatively slight degree of muscular spasm and absence of retraction of the head with a positive intradermal tuberculin test, are all very suggestive of tuberculous infection of the meninges.

Poliomyelitis is sometimes ushered in with mildly meningeal symptoms but hyperæsthesia is usually marked in the pre-paralytic stage and disparity in reflexes is another useful point of distinction. In this stage the deep reflexes are often unequally increased on the different sides just as later they are unequally decreased. In the pre-paralytic stage of poliomyelitis there is marked disinclination to flex the neck on the body on account of the pain experienced in doing so. In meningococcal meningitis, on the other hand, the patient is unable to flex the head on the neck because of the spasm of the neck muscles.

An examination of the cerebrospinal fluid (which should never be omitted) completes the differentiation between the two conditions. In poliomyelitis the fluid is clear, or if not has a "ground glass" appearance, with a moderate increase in lymphocytes, but there is no decrease in chlorides, and tubercle bacilli are absent.

One or two points in the treatment of cerebrospinal fever remain for consideration. The diet should be exceedingly liberal. Food must be forced on these children to counteract the excessive wasting which is a feature of the disease. Egg flips are a useful means to this end. The care of the skin is very important and if neglected bed sores are inevitable. The liberal painting of the lumbar region with iodine for puncture work is

	Meningeal Meningitis	Other Forms of Pyogenic Meningitis
Onset	May be sudden more commonly insidious in infants	Often sudden
Stiffness of neck	Marked	Present
Head retraction	Marked In later stages extreme with opisthotonus	If present slight and not progressive
Kernig's and Brudzinski's signs	Often present in infants—always in older children	Often present in infants—always in older children
Petechial rash	May be present	Absent
Herpes labialis	Not uncommon	Absent
Early paralysis	Uncommon	Uncommon.
Associated conditions	Generally some history of injury to head & fall	Some pneumococcal or streptococcal lesion e.g. lobar pneumonia pneumonia peritonitis otitis media mastoid disease septicaemia etc.
Wasting	Extreme in protracted cases	Not unduly marked
Spasticity of limbs	Very marked in chronic cases	Not unduly prominent
Hydrocephalus	Common in chronic cases	Rare
Cerebrospinal Fluid	Character	Turbid
	Cell content	Markedly increased
	Predominant cell	Polynuclears
	Organisms	Meningococci
	Other features	Sugar diminished or absent
		Sugar may be diminished.

## VI

Anterior Poliomyelitis	Epileptic Encephalitis	Tuberculous Meningitis
Often sudden	Often insidious	Generally insidious
Absent	Absent	Present
Absent	Absent	Absent
Absent	Absent	Often present in infants— always in older children
Absent	Absent	Absent
Absent	May occur	Absent
Flaccid paralysis of limbs very common	Uncommon	Uncommon except for squint and ptosis
Nil	Nil	Pre-existent tuberculous disease of bones joints glands or abdomen
Confined to affected limbs	Nil	Progressive never extreme
Absent	Severe in later stages of <i>some forms</i>	Not unduly prominent
Does not occur	Does not occur	Does not occur
Clear	Clear	Clear
Slightly increased	Increase slight or absent	Much increased
Lymphocytes	Lymphocytes	Lymphocytes
Nil	Nil	Tubercle bacilli
Sometimes fluid has "ground glass" appearance	Increase in sugar the most constant at normality	Chlorides always reduced

a certain means of producing bed-sores. A spot of iodine the size of a threepenny piece is quite sufficient. Alternatively the parts may be painted with 1 per cent picric acid solution or simply rubbed with ether meth. Icebags relieve headache and a bromide mixture helps also. The bladder and rectum have to be watched and if necessary artificially emptied.

Sulphanilamide preparations have been recently advocated for the treatment of bacterial meningitis. Tablets by the oral route may be given until the fluid is sterile, the dose being graded to suit the age of the child. In the more chronic stages of meningococcal meningitis (when a clear fluid is obtained with persistence of neck rigidity) I have found vaccines of service. An autogenous vaccine prepared from the organism isolated from the cerebrospinal fluid gives the best results, but the stock polyvalent vaccines are also very beneficial.

The essential points in the differential diagnosis of acute infections of the central nervous system are set out in Table VI.

### SMALLPOX AND VACCINATION

The practice of infant vaccination has shifted the age incidence of smallpox to adult life. Smallpox at any age has been unknown in Ireland for over thirty years and this feature in our epidemiological history is no doubt responsible for the increasing neglect of infant vaccination although the percentage of infants vaccinated in the Free State still remains considerably higher than in Great Britain. As to the wisdom of discontinuing a compulsory scheme of vaccination against smallpox a great deal might be said. It certainly does seem illogical that the law should force parents to have their children protected against a non-existent disease while allowing them perfect freedom of choice in relation to diphtheria which is *endemic*. *The whole position requires review in the light of existing conditions.* The substitution of compulsory immunisation against diphtheria for infant vaccination would be a decided gain to the public health but at the same time power should be given to local public health authorities to impose compulsory vaccination whenever the incidence of smallpox in their areas rendered such a step desirable.

## Vaccinia

Vaccinia is a disease caused by the intradermal inoculation of cowpox virus. The incubation period is three days. The eruptive lesions, which are usually confined to the traumatised area, begin as red raised papules, which become vesicles by the fifth day and are pustular by the eighth day. The fully developed lesions are ringed with a red areola. From the tenth day desiccation sets in and the lesions crust. Two or three weeks later the crust falls off. While the pocks are maturing general symptoms, *e.g.*, headache, malaise, insomnia and anorexia, may occur. Sometimes erythematous rashes appear. Very rarely a generalised papular eruption occurs in vaccinia, crops of papules appearing between the fourth and tenth day, each going through the usual stages of maturation. Sometimes the patient transfers vaccinal lesions to other parts of his body, accidentally, by auto inoculation, and the mother may similarly inoculate herself from her infant.

Vaccination should be performed with a minimum of trauma and with the same aseptic precautions as are adopted in minor operations. Failure to prepare the skin or sterilise the instruments results in cellulitis of the arm, which in young infants may lead to serious consequences. The skin of the arm should be rubbed with ether and then with spirit and the lymph then deposited on the cleansed area. A short prong, the "teeth" of which are not long enough to draw blood, is the best vaccination "lance". The "teeth" are pushed into the skin through the lymph and the handle is then turned through a half circle and the prong withdrawn. With the spatulate end of the lance the lymph is gently rubbed into the circular indentation produced, until the surface is dry. A small aseptic dressing is then applied. With this technique, no trouble of any kind has followed many thousands of vaccinations. One insertion is quite sufficient to produce an immunity lasting at the very least five years. In times of smallpox prevalence it would be desirable to revaccinate at the age of five years, before the child enters school. If maximal protection be desired—*e.g.*, in the case of infants going abroad—the traditional four insertions may be made. It is generally agreed that infants are immune to post vaccinal encephalitis, and, indeed, this fact constitutes the only valid argument in favour of the continuance of vaccination during the first year of life in this country.

### Post-vaccinal Encephalitis

Between the years 1923-29 some 400 cases of post vaccinal encephalitis occurred in Europe, mainly in Holland. Since then the reported cases have been very few.

The onset is sudden, very often on or about the eleventh day following a primary vaccination during adolescence. There is pyrexia, vomiting, headache and stupor or coma. Paresis of limbs may occur and Babinski's sign is almost invariably positive. Gradually deepening coma develops in roughly a third of the cases with death within a week or ten days of the onset of disease. Favourable cases recover after a few weeks and may be left with varying degrees of mental and physical impairment. The cerebrospinal fluid may be quite normal, but a slight increase in cells (lymphocytes) is usual. Repeated lumbar puncture is the most hopeful line of treatment. The cause of post vaccinal encephalitis is not precisely known, although many theories have been advanced to explain it. It is probable that its origin is similar to that of the encephalomyelitis which is met with occasionally after measles, chicken pox and other infectious diseases.

### ERYSIPELAS

Erysipelas is caused by the inoculation through the abraded cuticle of a hæmolytic streptococcus which is akin to the causative organism of scarlet fever.

#### Erysipelas Neonatorum

It is not infrequently met with in the new born as a consequence of umbilical sepsis. These cases are exceedingly fatal. The erysipelatous process consists of an intensely red area with a raised spreading edge and associated with marked constitutional symptoms. The temperature is often in the region of 104° or 105° and there is considerable toxæmia. In cases of umbilical origin the course is generally rapid, death occurring, with or without terminal hyperpyrexia and convulsions, in a few days. Bleb formation occurs over the erysipelatous area. Enlargement of the liver with jaundice may be seen also.

#### Erysipelas in Other Situations

In other situations erysipelas in infants differs from the disease as seen in older children and adults in its greater tendency to

migrate. An erysipelas beginning on the face often spreads over the scalp, down on to the back and lower limbs, and then advances over the front of the body from below upwards. It is noteworthy that the toxæmic symptoms in these cases are not nearly so marked as in the cases originating from umbilical sepsis. The risks of a fatal termination are, however, considerable in all cases. Migrating erysipelas may localise in certain situations—*e.g.*, a hand or a foot—and there cause cellulitis, which may go on rapidly to gangrene. There is the further risk at any time of the supervention of streptococcal bronchopneumonia. The case mortality of erysipelas in infants is never less than 30 per cent. Relapses are frequent and are frequently fatal.

In toxæmic cases the diet should be fluid, but in the protracted migrating cases the child must be given a liberal diet in spite of pyrexia. The most soothing and efficacious local application is white lint soaked in a saturated solution of magnesium sulphate, which should be kept constantly moist. Application of ice to the head relieves headache, allays restlessness and staves off convulsions. Brandy in small doses (3ss q.q.h.) is a good stimulant in erysipelas. Serum therapy should always be tried in toxæmic cases. Doses of 3–6 000 units of streptococcal antitoxin intramuscularly may be repeated twice or three times at intervals of twenty-four hours. Treatment with sulphamid preparations should be given in doses proportionate to the age both orally and intramuscularly. Quite young infants may be given 2 c.c. of prontosil intramuscularly once a day, and half a tablet of prontosil album crushed in milk three or four times a day can be prescribed at the same time. The drug should be discontinued when the disease is under control. Recent work has tended to show that the non-staining "album" preparation is a more efficient bactericide, even when orally administered, than the original prontosil rubrum.

An iron and strychnine tonic is indicated in convalescence. It is essential that the infant's upper limbs be restrained, for nose picking and face-scratching may initiate a relapse. A little cold cream on cracked nostrils often stops irritation, and the wearing of white cotton gloves during sleep is another safeguard against re-inoculation and relapse.

## CHAPTER XVII

W R F COLLIS

### THE HEART, VESSELS AND BLOOD DURING INFANCY

(Circulation of Blood during Fœtal Life Changes at Birth—Congenital Malformations Dextrocardia Coarctation of Aorta Patent Ductus Arteriosus Patent Septum Pulmonary Stenosis and Atresia—Malformations Great Vessels—Idiopathic Hypertrophy of Heart—Diagnosis and Prognosis—Examination of Heart during Infancy—Infective Disorders of Heart Pericarditis, Endocarditis (Acute and Subacute)—Diseases of Vessels—The Blood Primary Anæmia Acholuric Jaundice Racial Anæmias Secondary Anæmias Deficiency Anæmia, Infection and Intoxication—Treatment—Leukæmia )

If the various types of congenital heart disease are to be understood it is necessary to have a clear conception of the circulation of the blood in the latter part of fœtal life This is presented diagrammatically on p 179

If the diagram is studied it will be seen that in the fœtus oxygenated blood comes from the placenta by way of the umbilical vein This divides on entering the body one branch (the smaller) passing direct to the inferior vena cava through the ductus venosus the other joining the portal vein before it reaches the liver hence the blood from the inferior vena cava as it reaches the right auricle is mixed, being partly venous and partly oxygenated If the heart is examined it will be found that there is an opening from the right auricle to the left auricle *the foramen ovale*, and hence only some of the blood passes through the tricuspid valve into the right ventricle, the remainder going straight across through the foramen ovale into the left auricle thus less blood passes into the pulmonary artery in the fœtus than in the child after birth But even this limited amount is more than the lungs need in their unexpanded condition and there is another shunt, *the ductus arteriosus*, which joins the pulmonary artery and the aorta and takes the remaining overflow From the common iliacs the two hypogastric arteries go to the umbilicus and continue as the

umbilical arteries to the placenta, carrying the impure foetal blood there to be oxygenated

This general arrangement has the effect of making the foetal blood supply less oxygenated than the blood after birth, for not only is the blood less oxygenated by passage through the

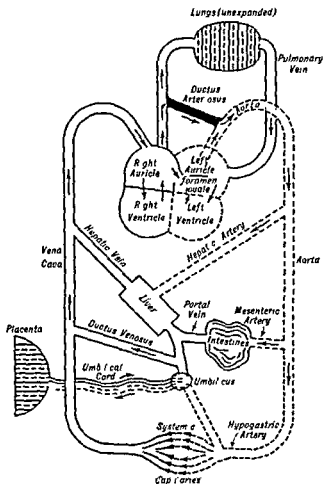


FIG. 16.—Diagram showing circulation of the blood during the latter part of foetal life

placenta than through the lungs, but no oxygenated blood is supplied to the foetal tissues till it has been diluted with venous blood. This is compensated for by the foetal circulation having a larger number of red blood corpuscles and more hemoglobin than is found in the child after the first week of life.

**Changes at Birth** Immediately after birth the placenta

ceases to be the child's respiratory organ and the lungs expand and take its place. When the blood ceases to pass through the umbilical arteries and vein these gradually atrophy till they are merely represented by fibrous bands though they remain partially open for several weeks after birth. The foramen ovale has a flap valve on the left side and as soon as the blood pressure in the left auricle becomes equal to that in the right the valve closes adhesions form and after some weeks it remains permanently closed. Similarly with the rise in the blood pressure in the aorta the ductus arteriosus ceases to function and undergoes atrophy. At the same time the blood passing through the lungs becomes more highly oxygenated than previously when it received its oxygen from the maternal blood in the placenta and no longer requires the same number of red blood corpuscles hence there is a hæmolytic of the surplus erythrocytes which produces a physiological jaundice in about 30 per cent of normal babies (see p. 11).

**Congenital Malformations of the Heart.** A great many malformations of the heart have been described occurring either together or singly. One of the most useful recent classifications is that given by O'Reilly who has simplified and shortened Abbott's original groupings—

**Group I. Acyanotic forms (without abnormal shunts or communications)** (a) *Dextrocardia*. In simple cases there may be no signs except the finding of the heart on the right side, the condition is however not uncommonly associated with other cardiac malformations.

(b) *Coarctation of the Aorta*. This consists essentially in a narrowing of the lumen of the aorta. The stenosis occurs usually in the neighbourhood of the orifice of the ductus arteriosus. If it occurs distal to the opening the ductus tends to remain patent. In certain exceptional cases its narrowing occurs proximal to the ductus.

It is very difficult to diagnose this condition in infancy. As the child grows older however certain definite signs make their appearance. There is great hypertrophy of the left ventricle. The pulse in the vessels arising below the constriction is smaller than in those from above and the blood pressure is less. Hence the blood pressure in the neck and arms tends to be higher than in the legs—sometimes it is impossible to obtain the arterial blood pressure in the popliteal space. As the child grows older compensatory collateral circulation

becomes established, and much enlargement of the dorsal scapular, internal mammary and intercostal arteries takes place. The former can be seen standing out and pulsating when these children are examined after they reach their seventh year. Erosion of the ribs by the communicating branches between the intercostal vessels sometimes occurs and notching of the ribs can be seen in the radiograph.

There is usually a systolic thrill to be felt over the upper portion of the cardiac area and pulsation can be seen in the suprasternal notch. A loud systolic murmur is heard two or three finger breadths to the left of the sternum in the third and fourth spaces.

The prognosis is often good and the condition is compatible with life to an old age though under development is common.

**Group II Potentially Cyanotic Forms** (Cases with an arterio venous shunt with or without reversal of flow)

(a) *Patent Ductus Arteriosus* This is not a malformation but merely a persistence of foetal circulation due as a rule to some other cardiac defect. It is commonly associated with pulmonary stenosis or coarctation of the aorta. Its characteristic sign is a continuous murmur (the humming top or machinery murmur) harsh during systole and tailing off somewhat in diastole.

Radiographs often show a characteristic shadow just above the base of the heart on the left side. It is caused by hypertrophy of the pulmonary artery.

(b) *Defects in Auriculo ventricular Septa* Patent foramen ovale is of little importance in an otherwise normal heart. It produces no symptoms and will not be recognised during life. The condition is however often associated with other congenital abnormalities of the heart when it may give rise to a loud murmur. *Defects in the interventricular septum* are among the commonest congenital malformations and when unassociated with other abnormalities can often be diagnosed during life. The opening is usually high up in the septum and varies greatly in size. In extreme cases the septum is almost entirely absent and the heart is converted into a three chamber organ. Heart block is an occasional though rare complication.

Due to pressure being higher in the left ventricle than in the right the blood flows from left to right with each contraction of the heart causing a loud systolic murmur heard all over the front of the heart and being associated not uncommonly

with a marked thrill. There may be some enlargement of the heart, but this is never great and there are seldom any general symptoms except when the condition is associated with other cardiac abnormalities. The diagnosis rests upon finding the above signs in the absence of symptoms of morbus cordis.

**Group III Cyanotic Forms** (Cases of veno arterial shunt)

(a) *Pulmonary Stenosis and Atresia*. This malformation is probably the best known of these conditions due to the physical signs which tend to be associated with it—intense cyanosis, clubbing of the fingers and polycythæmia.

The lesion may be in the conus arteriosus at the pulmonary valve or in the pulmonary artery. Except in very slight cases compensation by way of interventricular communications and a patent ductus arteriosus is necessary for life. In certain cases this compensation is almost perfect and the patient can live a normal life. In others this is far from being the case, and he exhibits marked cyanosis: the lips and gums are purple, the latter bleeding easily, the veins of the eyes are prominent and the skin gradually assumes a dusky bluish colour. Infants appear deathly pale with blue lips. As time goes on the fingers become clubbed and a marked hypertrophy of the blood takes place. Sometimes the polycythæmia reaches ten million red blood corpuscles per cubic millimetre; more commonly it is about seven million. The cyanosis is due to failure of the pulmonary blood supply and lack of oxygenation combined with a mixing of the arterial and venous blood supplies by the associated shunts.

The most characteristic sign is a loud, harsh systolic murmur heard best in the second and third left interspaces, conducted up and out towards the clavicle and often heard behind at the angle of the scapula. A systolic thrill is also usually present. The heart will be enlarged to the *right* but not to the left.

The prognosis depends upon the degree of stenosis and the associated lesions. Severe cases die shortly after birth, moderate cases are compatible with life but are associated with much cyanosis and patients who survive infancy and childhood form the well known 'blue man' type seen in all general hospitals. Mild cases give rise to few signs and lead to little invalidism. Many other lesions have been described associated with pulmonary stenosis such as patent septum or patent foramen ovale, transposition of the arterial trunks and the well known tetralogy of Fallot (*e.g.* pulmonary stenosis,

septal defect dextro position of the aorta marked hypertrophy of the right ventricle)

*Malformations of the great vessels* are not uncommon and are very variable. The pulmonary artery and aorta may be partially fused or transposed. In the latter case the aorta may merely pass in front of the pulmonary artery being still attached to the left ventricle or it may take its origin from the right ventricle.

The symptoms and signs of these conditions are variable and can seldom be diagnosed with any accuracy. Cyanosis without an associated murmur is said to be suggestive of arterial malformation.

*Idiopathic hypertrophy of the heart* is a rare condition (we have only seen one case) though probably most cases pass undiagnosed. The cause is obscure. The main sign is hypertrophy of the cardiac muscle unassociated with any valvular lesion or cardiac malformation. Diagnosis can be made by percussion of the heart which will be found much enlarged to the left and an x ray will show a very large heart shadow. The condition appears always to be fatal death being due to circulatory failure.

**Diagnosis and Prognosis** During infancy it is unwise to attempt to make a more definite diagnosis than that of congenital heart disease. Later on as the signs and symptoms become clearer it may be possible to differentiate such conditions as coarctation of the aorta pulmonary stenosis and uncomplicated septal defect but before the end of the first year the physician is wise to be conservative. He should always give a grave prognosis for many cases die suddenly of syncope.

Even the general diagnosis of congenital heart disease is not always easy particularly during the neonatal period. At this period attacks of cyanosis also not uncommonly occur associated with respiratory conditions particularly atelectasis and cerebral birth trauma. In those cases where a murmur is heard the diagnosis is definite but when this is absent it may be impossible.

In later infancy the condition has to be differentiated from infective endocarditis and those states associated with functional cardiac murmurs. Infective endocarditis is extremely rare during infancy but may occur at any age and has even been reported in the *fœtus*. It is usually accompanied by a swinging temperature and signs of septicæmia.

*Functional murmurs* are caused by a number of conditions. Sometimes the heart presses upon the lung and the murmur is cardio-respiratory. When the position of the child is changed or when he holds his breath the murmur disappears. In severe anemia a *hæmic murmur* may be heard in certain cases. Sometimes there is no explanation for a small localised murmur. These murmurs give rise to no symptoms and tend to clear up as the child grows older.

**Treatment.** There is no specific treatment for congenital *morbus cordis*. The infant should be protected from all respiratory infection as this is apt to embarrass the right side of the heart. If the child survives infancy he must be taught to live within his capacity. The tendency in later life to develop infective endocarditis should be guarded against as much as possible by special attention being given to septic foci such as infected teeth and tonsils.

### Examination of the Heart During Infancy

**The Pulse.** The pulse during infancy is both faster and more variable than in later life and varies between 100–150 beats per minute. At six months of age it should be about 110 per minute. In small infants an irregular heart beat need not cause anxiety as it is a common finding in normal children at this age. The examination of the heart during infancy is always difficult and it becomes impossible if the child is crying. So here again as when examining the lungs or the abdomen the baby should be given a bottle during the examination. Percussion must be light and it is sometimes better merely to tap the middle finger of the right hand on the chest than to attempt percussion in the usual way. Under one year the left border of the heart should reach just outside the left mammary line and the apex beat should be in the fourth interspace. It is often difficult to follow the classical method of examination—inspection, palpation, percussion and auscultation—when dealing with a small baby whom it may be unwise to undress completely. Nevertheless the physician is wise to follow the old routine as closely as possible else he will tend to miss important signs. Above all, the children's physician needs patience, gentleness and care. Heavy percussion is valueless and cold hands will make the baby cry and struggle.

### Acute Infective Disorders of the Heart During Infancy

**Acute Pericarditis** This is a very rare condition to find during infancy. It occurs as a complication of some other severe infection and is mentioned again in connection with pneumococcal empyema. Hemolytic streptococcal septicemia and staphylococcal and tuberculous infections also occasionally cause pericarditis during infancy. The diagnosis is very difficult and is rarely made during life. Signs and symptoms may be almost absent, the baby cannot complain of pain and friction rubs are often absent while the degree of enlargement of the cardiac dullness may be difficult to detect. In our experience an x ray is the best method of diagnosis in these cases. It will show a general enlargement of the cardiac shadow with a specially typical globular increase to the right of the sternum. Pericarditis should always be kept in mind when some infective state such as empyema continues to cause a high temperature and prostration after competent drainage has been established and the primary condition appears to be improving.

**Acute Infective Endocarditis** This is another condition only occasionally met with during infancy. It may complicate some acute infection, such as erysipelas or umbilical infection in the new born the infecting organism being usually the hemolytic streptococcus or the staphylococcus. The symptoms of septicemia may overshadow the local heart signs and the diagnosis only be made at autopsy.

### Diseases of the Vessels

The only condition of any interest appearing under this heading during this age period is *arteriosclerosis* which has been reported in the new born. The condition is similar to that seen in adults—tortuous arteries, containing atheromatous plaques.

Recently much attention has been called to a condition of calcification of the media of the arterial walls supposed to be due to a hyper vitaminosis D. More work requires to be done, however, before this is accepted, as the condition may occur in certain cases receiving a given dose of vitamin D, and not in others on the same dose. It is not improbable that the parathyroids may also play a part in its causation.

## THE BLOOD

## Primary Anæmia

In the section dealing with pathological conditions in the new born icterus gravis neonatorum was described (see p 51) A number of other primary anæmias need mention here The usual picture presented by these hæmolytic anæmias unassociated with jaundice is as follows —

At birth the hæmoglobin is approximately 100 per cent and the baby appears normal Almost at once however he begins to go down hill becomes pale but does not develop jaundice Then he commences to refuse nourishment later to vomit and often dies In these cases little regeneration is found in the blood and the bone marrow is aplastic In some cases the child goes down hill steadily and dies in others if he is tided over the first few weeks regeneration takes place with complete recovery The only effective treatment is transfusion which should always be undertaken as soon as the condition is diagnosed It will often save life

Certain rare hypoplastic and aplastic anæmias occur also after the neo natal period Their cause is obscure and their treatment unsatisfactory

Acholic or familial jaundice is a true hæmolytic anemia which occurs at any age It is characterised by recurrent attacks of jaundice enlargement of the spleen and increased fragility of the red blood corpuscles The latter is the distinguishing characteristic upon which the diagnosis often rests In acholic jaundice if the fragility of the erythrocytes is tested hæmolysis will be found to commence between 0·6–0·7 per cent saline (normal fragility being 0·4 per cent approximately) Periodic attacks of hæmolysis of the red blood corpuscles occur associated with jaundice of variable severity This is followed by increased hæmatopoietic activity great numbers of reticulocytes and a fair number of normoblasts appearing in the circulation The disease may be congenital or acquired It has a tendency to be hereditary and tends to occur in other children of the same family It may commence during early infancy or in later life Sometimes it may be confused with icterus gravis neonatorum though we have never encountered it during the first week of life

The most effective treatment for the condition is splenectomy which improves or cures the majority of cases

The condition is rare during infancy and students are referred to the text books on general medicine for fuller details

### Racial Anæmias

Certain special types of primary anæmia have been described in certain races. Sickie celled anæmia is a somewhat analogous condition to acholuric jaundice and is characterised by the peculiar shape of the erythrocytes denoted by the name it occurs only in negroes. Cooley's anæmia is another primary congenital anæmia progressive in type and resistant to treatment, met with only in the Mediterranean races

## SECONDARY ANÆMIAS

### Deficiency Anæmia

This group may be defined as anæmia due to lack of some essential hematopoietic factor. These substances may be exogenous supplied in the food or endogenous and manufactured in the body. Of recent years our knowledge of them has greatly increased due to the work of Mc Kay, Josephs, Parsons and his co-workers, Witts, Murphy and others though their whole mode of interaction is still obscure in many ways.

Witts claims that the following list of substances are required for hematopoiesis —

Liver substance

Vitamin B

Iron and copper

Vitamin C

Thyroxin

And that lack of liver substance and vitamin B produce the megalocytic type of anæmia (*e.g.* pernicious anæmia)

Lack of iron and copper, vitamin C and thyroxin on the other hand, produce the hypochromic microcytic type. We are only concerned with the latter here as pernicious anæmia is unknown during infancy.

Deficiency of iron in the infant's diet is by far the commonest cause of anæmia in the baby. This is due to the fact that milk (both human and bovine) does not contain enough iron for the needs of the average baby after he is six months old. This need will be felt by the child very much sooner if he has a poor supply

of iron in his liver at birth due to the mother having had a deficient diet during her pregnancy or if he suffers from some infection or debilitating disease. Hence infants fed on milk alone for over six months show as a rule some degree of anæmia. This is particularly noticeable in artificially fed babies.

The part played by copper in the cure of anæmia is an interesting one. In the old days iron therapy cured this form of anæmia. Then came the craze for purifying everything and the iron administered as medicine to children was given in the pure form. The new preparation failed to cure the anæmia. Research revealed that the old impure preparation of iron contained small amounts of copper and that without this impurity iron administration was valueless as a cure for anæmia. Now all medicinal iron preparations contain small quantities of copper. Exactly how the copper acts is not clear. Its action has been compared to that of a catalyst.

Vitamin C deficiency causes scurvy and one of the main symptoms of scurvy is hæmorrhage. Hence it is not unnatural that scurvy should be associated with a profound anæmia. Vitamin C also plays a part in the maturation of the red blood corpuscle. This is apparent in subscorbutic conditions for before the appearance of the actual hæmorrhagic symptoms of scurvy an anæmia appears in the child which is cured only when vitamin C and iron are added to the diet.

Thyroxin also plays an essential role in the formation of the red blood corpuscle. Witts and others have described a form of microcytic anæmia in adults which resisted all ordinary forms of therapy but responded at once to small doses of thyroxin. Definite hypothyroidism either in the child or the adult is commonly associated with a degree of anæmia. Our recent work on the effect of thyroxin in cases of prematurity suggests that thyroxin may be an important factor also at this early age though further work is required to establish this hypothesis. In the treatment of anæmia therefore thyroxin should never be forgotten.

**Infection and Intoxication.** Acute infections (pyogenic) and chronic infections (syphilis, tuberculosis, etc.) may cause the rapid appearance of anæmia in infants from the first few days of life onwards. Whether the infection destroys the blood directly or checks growth by intoxication of the bone marrow it is difficult to say. The latter is the most probable. Some

times, however, the appearance of anemia is so rapid that definite breakdown of the blood appears probable. Anemia of infective origin is very commonly associated with the deficiency types of anemia described above.

**Treatment** These forms of anemia can be prevented by proper diet. This can only be done, however, in large cities with the help of the infant welfare centres and by the spread of the knowledge of dietetics. It is absurd that in a world where there appears to be a surplus of foodstuffs of all kinds, so that in many places they are actually being destroyed, many of our children should still be on a deficiency diet. It is necessary to stress this point because it is waste of time, on the part of the doctor, telling a mother to give extra milk, orange juice and egg yolk to her anæmic baby when she has not the money to procure these necessary foodstuffs.

The system which we adopt generally to prevent nutritional anemia has already been described in the chapter dealing with infant feeding. It consists essentially in assuring that the baby shall receive the necessary quantity of milk and vitamins till he reaches a weight of 15 lb. or is six months old, when additions are made to the diet.

The treatment of anemia, once established in the baby, depends upon its degree and the age of the child. In the early stages the administration of a teaspoonful of egg yolk once a day to a balanced diet will often be sufficient to restore the blood to normal in a short time. In severer cases this will not be sufficient, and it is necessary to give large doses of iron. The general principle is undoubtedly to give iron in as massive doses as possible so as to stimulate production of erythrocytes. The difficulty is, however, that small babies do not tolerate iron at all well. During the second half of the first year the problem is much simpler as the baby by now will be able to take iron without ill effects. During the first few weeks, particularly in the cases of prematurity, even the smallest amounts of iron are apt to produce loose motions and sore buttocks. Each case has therefore to be treated on its own merits. The general principle is to give the largest amount of iron that can be tolerated. For this purpose reduced iron, gr  $\frac{1}{2}$ -1, may be given twice a day to begin with, or ferri et ammon cit, gr 1-v. It is always well to combine these with egg yolk, which not only contains iron itself, but is also rich in vitamins. Orange juice even in very small quantities should

always be given and thyroxin administered cautiously to premature and weakly infants

Ferrous sulphate	gr 1½
Dilute hypophosphorous acid	℥ ½
Dextrose	gr 15
Aq chloroformo	ad 5 i

Recently Dr Helen McKay with the above mixture reports very successful results which the author is able to confirm. The ferrous sulphate retains its potency in this mixture for a considerable period of time. The dose must be varied to suit the age and tolerance of the infant but ℥i-xx can be given to the new born baby and gradually increased till at six months old the child may be given 1 drachm three times a day.

### Leukæmia

Leukæmia is met with at any age, being seen occasionally during infancy. The main characteristics of the disease are dealt with in all general medical text books and hence need only a brief mention here.

There are two main types lymphoid in which the lymphoid tissue and lymphocytes are affected and myeloid which involves the myeloid cells. The former is the commonest in childhood and the only type apt to cause confusion in diagnosis.

Myeloid leukæmia is diagnosed by the rapid and great enlargement of the spleen and the characteristic appearance of the blood, which shows numerous myeloblasts and myelocytes.

Lymphatic leukæmia during infancy often runs a very acute course, simulating an acute infection. The onset is often abrupt, being associated with vomiting, fever and collapse, and the child may die in a few weeks. There is usually swelling of the lymph glands, most marked in the cervical region, but also in the groin, axilla and elsewhere. Sometimes the glands do not enlarge. The spleen is often greatly enlarged. The parotids, *submaxillary* and *lacrymal* glands may become infiltrated with leukæmic tissue.

Hæmorrhage under the skin or from the mucous membranes commonly occurs in childhood. The gums may become swollen and hæmorrhagic, somewhat resembling their condition in scurvy. The white cells may either show a leucopenia or a leucocytosis. When the former occurs the general condition

is called aleukæmic leukæmia. Lymphocytes are found to predominate, being usually between 90-98 per cent of the total leucocyte count. They are often larger than normal, irregular in outline and show abnormally staining nuclei. A marked anemia of the erythrocytes is usually present, the red cells numbering between 1 000 000-3,000 000. The outlook is hopeless, death always taking place and no treatment being of any avail.

The diagnosis can be made from other acute infections with similar symptoms by the examination of the blood. Confusion is only likely to occur in cases of *glandular fever* (infectious mononucleosis). In the latter condition it is not uncommon to find fever, glandular swellings and a lymphocytosis in which many of the lymphocytes are immature. Glandular fever is infectious and tends to occur in epidemics. Sometimes in these cases only the subsequent course of the disease will give the diagnosis, as glandular fever is as benign as leukæmia is fatal. We have seen a case of a baby girl aged eight months in whom the diagnosis remained in doubt for two months, during which period she ran an irregular temperature, showed fluctuating enlargements of the cervical glands and a lymphocytosis, and then made an uninterrupted recovery.

## CHAPTER XVIII

W R F COLLIS

### BRONCHITIS AND PNEUMONIA

(Immunology Bronchitis Pathology Symptoms Diagnosis Complications Prognosis Treatment Lobular Pneumonia Pathology Signs Symptoms and Diagnosis Complications—Lobar Pneumonia Pathology Signs and Symptoms Diagnosis Prognosis Complications Treatment—Interstitial Pneumonia Pathology Signs Symptoms and Diagnosis Prognosis Treatment)

#### Bronchitis Lobular (Broncho) and Lobar Pneumonia

THESE three conditions are among the commonest diseases found in infancy and childhood and together account for a large number of deaths per annum. All three are associated with the same invading organism the pneumococcus though bronchitis is often caused by other organisms as well. The pneumococcus is always present in lobar and lobular pneumonia though other organisms such as Pfeiffer's bacillus or a streptococcus may also be present. There is no definite explanation as to how such completely different syndromes may be brought about by the same invading germ. It has been suggested that bronchitis and lobular pneumonia are caused by the gradual spread of the infection down the respiratory passages while lobar pneumonia is essentially a blood stream infection. Certainly it is much more common to obtain a positive blood culture in lobar than lobular pneumonia.

All three conditions are much more frequently seen in the poorer classes than among the well to-do and all three occur in winter rather than in summer. These facts suggest that —

(1) Infection from over crowding is a potent factor here as with other droplet infections

(2) Under nourishment is a predisposing cause

The latter is borne out by the old observation that bronchitis and pneumonia are very often associated with rickets. The older observers were wont to describe respiratory and alimentary infections as part of the rachitic syndrome. It is doubtful if vitamin D plays any part in defence against infection but it is now well established that vitamin A is essential for the health of the mucous membranes. This prob-

ably explains the bronchitic tendency in rachitic children. Individual constitution (diathesis) is certainly an important factor in the ætiology. Certain families are particularly prone to respiratory infection while others appear almost immune. This may be due to some purely structural characteristic such as the shape of the larynx, or to the immunological response of their tissues. Mongols are much more prone to bronchitis and pneumonia than normal children and usually succumb to some such infection in the end.

Again children are more prone to bronchitis than adults. It has been suggested that this is due to the relatively small size of the bronchial tree in infancy and the tendency for secretions to become lodged in the bronchi instead of being coughed up. Also that the pulmonary artery is relatively bigger in early life than later and hence that the lungs are more liable to hyperæmic conditions at this age. In the author's opinion the liability to these infections in early life is due to lack of general immunity to infecting organisms. Immunity is a very complicated process many factors playing a part one of the most difficult to explain being allergy or hypersensitivity of the body to certain products of the germ. In early infancy lobar pneumonia is very often fatal from twelve to eighteen months onwards during childhood uncomplicated lobar pneumonia is almost benign and seldom causes death while in adult life it is one of the most fatal diseases. The explanation of this would seem to be that in infancy the invasive power of the pneumococcus meets with little resistance in childhood a degree of resistance has been established while in adult life although a resistance to the invasive power of the organism is considerable and complications (such as otitis empyema etc.) much rarer than in infancy and childhood the patient has become allergic (hypersensitive) to certain products of the pneumococcus which produce the characteristic toxic phenomena of adult pneumonia.

### Bronchitis

This is probably the commonest infection during the first few years of life. Still's figures show that it is twice as common during the first year of life as the second.

**Pathology** Bronchitis due to pyogenic infections is a diffuse process affecting the bronchial tubes of both lungs. When

examination reveals a patch where localised bronchitic sounds are heard in one lung only tuberculous infection should always be suspected

In older children the infection seldom extends beyond the larger tubes but in infancy the small bronchi are not uncommonly infected leading to the serious condition of capillary bronchitis. The mucous membrane undergoes a catarrhal inflammation passing through the usual stages to the exudative state when purulent mucus is coughed up or absorbed. In children the exudate may contain much fibrin and may become tenacious and difficult to dislodge from the smaller tubes. In all severe cases some degree of emphysema occurs. Sometimes it is very marked and the cardiac dullness almost disappears.

**Symptoms** *The symptoms and signs are very variable.* In mild cases slight elevation of temperature ( $99.6^{\circ}$  to  $100^{\circ}$  F) slight increase in the respiratory rate and rhonchi heard over both lungs may be all and recovery may follow after a few days during which the infant is little distressed. In severe cases the onset may be acute the temperature rising rapidly to  $102^{\circ}$  F the child appearing flushed and cyanotic and coarse or fine râles being heard over the lungs while the respirations sometimes may reach sixty per minute. Partial pulmonary collapse very readily occurs in infants and usually accounts for some of the symptoms. Emphysema may develop rapidly with loss of cardiac and hepatic dullness. Cough is always present and is very variable in intensity. Acute bronchitis seldom lasts for more than two or three days the temperature and general symptoms then tending to subside. It may take a considerable time however for the condition to clear up completely and chronic bronchitis characterised by cough and on auscultation coarse râles may continue for weeks in certain cases.

**Diagnosis** In mild cases the diagnosis presents no difficulty. The child clearly is not severely ill the respirations though increased are not rapid the alæ nasi are not working and the inexperienced student will be able to make the correct diagnosis even if the baby is crying and the physical signs indistinct. In severe cases on the other hand diagnosis may be extremely difficult. Here let us say that the examination of the infant and young child requires special technique. It is quite useless

to attempt to elicit physical signs when the baby is crying and struggling, a state of affairs which will almost always occur if he is undressed and then hastily percussed and auscultated. In small infants a bottle should always be given to the baby while the examination is proceeding, in older children sweets are invaluable as an aid to diagnosis. The examination itself must be rapid, percussion must be gentle, the hands of the physician being warm and the nozzle of the stethoscope sufficiently small to fit closely to the chest wall. The main problem in the diagnosis of a severe case of bronchitis is whether any lobular pneumonia is present as well. The diagnosis turns on the severity of the symptoms as a whole and the finding or not of areas of consolidation. In severe cases the most experienced physician will often be doubtful of his diagnosis.

Asthma is very rarely met with in the age period covered by this book, and when it does occur is rarely diagnosed. The essential feature of asthma is expiratory difficulty, expiration being accompanied by a characteristic wheezing sound which, when once recognised, is unlikely to be mistaken for any other sign in future.

**Complications.** The commonest complication is direct spread up the large tubes into the bronchioles and thence into the alveoli with resulting lobular pneumonia. The organism (particularly the pneumococcus) may spread in many directions (though less commonly in bronchitis than in lobular pneumonia) and otitis, sinusitis, arthritis, meningitis, etc., may follow an attack of bronchitis. Apart from these complications due to the direct spread of the organism gastro enteritis is very commonly associated in infancy with bronchitis, being due partly to the swallowing of infected sputum and partly to the general toxæmia (see p. 105).

**Prognosis.** In simple uncomplicated bronchitis the prognosis is good at all ages. Severe bronchitis when associated with complications, particularly capillary bronchitis, on the other hand, has a very serious prognosis in infants under six months of age.

**Treatment.** Prevention is the most important factor here. Balanced feeding with careful addition of the fat soluble vitamins, isolation from infection, avoidance of chill (particularly cold extremities in infants) and exposure to fogs, irritating dusts and east winds, will greatly reduce the chances of an attack. In acute bronchitis, as in laryngitis, cold or foggy

air must be excluded from the sick infant's room. In cold or foggy weather it is best to close the windows and open the door. In warm summer weather however these cases are best nursed in the open air if possible or in a room with the window wide open. The old remedy for bronchitis of the half tent combined with a steam kettle containing a pint of water to which a teaspoonful of tinct. benzoin co. has been added is very useful in the early stages when the cough is hard and troublesome. The temperature of the air around the child should not be allowed to rise above 65° F. and gas and electric stoves are bad as they overheat and dry the air.

Both expectorant and sedative drugs can usually be dispensed with in infancy and are to be avoided if possible. Where spasm of the bronchi is associated with mucus secretion a mixture containing potassium iodide gr.  $\frac{1}{4}$ — $\frac{1}{2}$  and tinc. stramonium ℥i ii may be given to a child of six months. A similar mixture is very useful if the bronchitis becomes chronic.

#### Capillary Bronchitis

This is a rare condition and is seldom uncomplicated by some degree of lobular pneumonia from which in young infants it is very difficult to distinguish during life. The onset is acute and is associated with cough, rapid respirations, fever usually between 100°–102° F. with varying degrees of prostration and cyanosis. The respirations are very rapid, sometimes reaching 80 per minute. Fine rales and crepitations are heard over both lungs while the ordinary sounds of respiration become faint. Resonance becomes exaggerated due to emphysema while no evidence of consolidation can be found. Death usually takes place on the fourth or fifth day in young infants. In non-fatal cases improvement often occurs rapidly from the third day and complete recovery may take place within a week.

The treatment of capillary bronchitis is similar to that of lobular pneumonia (see below).

### Lobular or Broncho Pneumonia

Lobular pneumonia may occur with sudden onset as a primary infection or gradually as an extension to the alveoli from the bronchi. It is often associated with infectious fevers such as measles, whooping cough and influenza. These conditions lower the general body resistance and allow the germ to propagate in the lung.

Lobular pneumonia together with gastro-enteritis with which it is often associated are the two commonest causes of death in young infants.

**Pathology** In most cases the lesions are found scattered throughout both lungs, the lower lobes being more particularly affected. If the baby dies in the first twenty-four to forty-eight hours little can be seen by the naked eye when the lungs are exposed at autopsy. The lower lobes may appear dark coloured and congested. Most of the lung can be inflated and will float in water. On section congestion and œdema will be found. Microscopic section only reveals the true pathology. There is much hyperæmia of the blood vessels and in these very acute cases it is not uncommon to find hæmorrhages under the pleura. The essential lesion which differentiates the condition from acute bronchitis however is that in lobular pneumonia areas are found where the alveoli are consolidated with red blood corpuscles and epithelioid debris. Usually the mucous membrane of the bronchi also shows catarrhal inflammation. If the disease has progressed for several days before the child dies these areas of consolidation are much more marked. The lung may present the classical red, white and blue appearance—the red areas signifying consolidation, the white emphysema and the blue collapse. Sometimes the consolidation is so great as to give the appearance of a completely consolidated lobe such as is met with in lobar pneumonia.

**Signs, Symptoms and Diagnosis** The classical signs and symptoms of lobular or broncho pneumonia may be tabulated as follows —

(1) *Respiratory Distress* The respiration rate is greatly increased, often being as fast as 60 per minute. The *alæ nasi*

are seen to be working and the accessory muscles of respiration are often called into play. The child appears restless and distressed, often cyanosed with blue lips.

(2) *Temperature* There is always high fever, usually of the swinging intermittent type, varying between normal in the morning to  $104^{\circ}\text{F}$  at night, though every type of abnormal temperature chart may be met with. In the same case the four hourly chart may show the intermittent type, while the twelve hourly has the unbroken high curve of lobar pneumonia.

(3) *Percussion* Usually it is difficult to make out definite areas of dullness though often it will be possible to observe that certain areas are more resonant than others.

(4) *Auscultation* Scattered areas will be found over which fine râles and high pitched or bronchial breathing will be heard.

In a typical case the diagnosis is simple but sometimes the course of the disease is very irregular and it is only by a careful study of all the symptoms together with the x ray appearances, that the diagnosis can be made.

Caseous pneumonia due to the tubercle bacillus, may present very similar physical signs. The temperature chart and x ray of the chest are also similar in the two conditions, and in a very sick child the tuberculin reaction is of little use, as it is not uncommonly negative whether the child is tuberculous or not.

*Prognosis* The outlook in lobular pneumonia is always grave under a year over 60 per cent of cases die, under six months the percentage is still higher. During the neo natal period it not uncommonly complicates atelectasis (see p 44), and in these cases usually leads to a rapid fatal termination.

In ascertaining the prognosis in any one case, more emphasis should be laid on the general condition of the patient and the presence or absence of complications especially gastro enteritis than upon the temperature. Often children with a swinging temperature reaching  $105^{\circ}\text{F}$  recover, while others die though their temperature has been normal for several days. As long as the child's general condition is well maintained and he continues to take his feeds there need be no immediate anxiety, but if diarrhoea supervenes and he refuses nourishment, the outlook rapidly becomes very grave. However, hope should never be abandoned or strenuous efforts at treatment relaxed till the child actually dies, as cases which

may appear almost hopeless to the physician not uncommonly suddenly take a turn for the better and recover

**Complications** General blood stream infection is less common in lobular than in lobar pneumonia. Hence meningitis, empyema and pericarditis are less commonly seen complicating the lobular type. *Otitis media* due to the spread of the organism up the Eustachian tube from the naso-pharynx is a more common complication.

Again gastro-enteritis is a common and deadly complication.

**Treatment** The treatment of lobular pneumonia is given with that of lobar pneumonia on p. 205.

### Lobar Pneumonia

Lobar pneumonia may occur at any age though it is not often met with in babies till after the first year. It occurs most frequently in the first three months of the year though cases may occur at any time.

It does not follow other debilitating diseases or appear as a spread from bronchitis but usually occurs suddenly in apparently healthy children. One attack does not confer prolonged immunity and repeated attacks are often seen. Not uncommonly one lobe after another may be attacked the previous consolidation clearing up as a fresh area commences.

The bacteriology differs little from that of adult lobar pneumonia the usual organism being one of the types of pneumococcus.

**Pathology** The general pathology also resembles that of adult lobar pneumonia the lung tissue passing through the stages of congestion red hepatisation grey hepatisation and resolution. In older children the appearance differs in no way from that found at autopsy in adults. In infants however it is rare to find a whole lobe involved more commonly the consolidation is found restricted to the apical region or to the base. Also in young infants the whole lung rarely presents a uniform appearance at autopsy. When cut across patches of normal lung may be found interspersed with patches of consolidated and congested lung in all stages from simple congestion to grey hepatisation. Indeed it is sometimes difficult to tell at autopsy whether the case is one of confluent lobular pneumonia or true lobar pneumonia.

One of the most important points in treating cases of lobar pneumonia is to remember its tendency to complications and to be on the look-out for them (see p. 204).

A chloride retention is usually found during the height of the fever together with a lowering of the alkaline reserve. Ketosis is often present and should always be looked for and treated.

The blood shows a high polymorphonuclear leucocytosis as a rule the count often rising to between 20 000-40 000 per cubic centimetre. It may rise even higher without the formation of localised pus. Hence a white cell count is of little value if only done in the later stages of the disease when an empyema may be suspected though a negative finding in a doubtful case

will be good evidence against the diagnosis of empyema. Blood cultures are frequently positive in children suffering from lobar pneumonia though less often than in adults. Marked bacteræmia is a bad prognostic sign.

**Signs and Symptoms.** Lobar pneumonia in childhood presents an altogether different clinical syndrome to the adult type. In children over one year there is seldom much prostration whereas in later life the patient as a rule appears much more prostrated than the degree of consolidation would suggest. Indeed it is not uncommon to see a child sitting up in bed playing with his toys while suffering from a patch of lobar pneumonia and a temperature of  $104^{\circ}\text{F}$ . In small infants however there seems little resistance to the invasive power of the organism the disease tends to spread the baby becoming increasingly cyanosed and distressed.

For the sake of clarity certain of the symptoms are tabulated below —

(1) *Temperature.* As in adult lobar pneumonia the temperature tends to be the high maintained type with only small fluctuations in contrast to the intermittent swinging temperature of lobular pneumonia (see Figs 17 and 19).

Fig 17 Lobar pneumonia showing typical crisis

Fig 18 Lobar pneumonia with pseudo crisis

Fig 19 Lobular or broncho pneumonia

The onset of the fever is sudden the temperature reaching  $104^{\circ}$ – $105^{\circ}\text{F}$  in a few hours and is maintained at this level for five to nine days as a rule when a crisis occurs the temperature either falling gradually by lysis or abruptly. Commonly there is first a pseudo crisis the temperature falling but the pulse and respiration remaining increased and the general condition unchanged. In these cases the temperature subsequently rises again after a few hours though seldom to quite the same height, it remains high for another twelve to forty eight hours after which if the baby survives a real crisis occurs the temperature falling and remaining normal or subnormal while the pulse and respiration rates decrease and the general symptoms of prostration subside (see Fig 18). Occasionally after a few days another lobe becomes involved and the temperature rises once more and the whole process is again repeated.

(2) *Onset.* The onset is usually abrupt the temperature rising in a few hours to  $104^{\circ}\text{F}$ . Often it is associated with

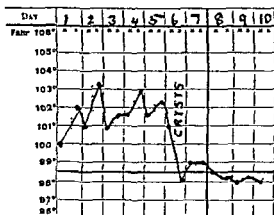


FIG 17—Chart from case of lobar pneumonia showing crisis

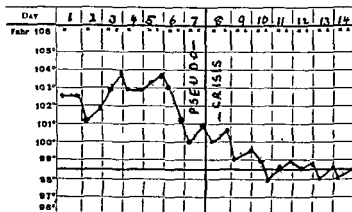


FIG 18—Chart from case of lobar pneumonia showing pseudo-crisis

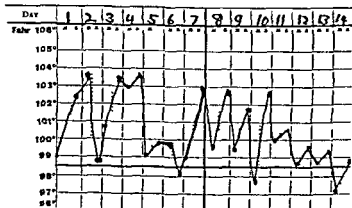


FIG 19—Chart from case of lobular or broncho pneumonia.

vomiting sometimes convulsions and not uncommonly with referred abdominal pain simulating appendicitis. Rigor as an early symptom is much rarer in children than adults, and seldom if ever seen under one year.

(3) *Pain* In the infant it is difficult to detect abdominal pain, but in older children a basal pleurisy with referred pain is common.

(4) *Respiration* This is always quickened. The normal pulse respiration ratio is usually changed. There is often a characteristic grunt associated with a short pause between inspiration and expiration. The alæ nasi can be seen working and the auxiliary muscles of respiration increase the respiratory excursion. The sputum is scanty and usually swallowed by small children. Cough is seldom a trying symptom and can usually be controlled by mild sedatives.

In certain cases there are marked meningitic symptoms (e.g., stiffness of the back, head retraction and a positive König's sign). Lumbar puncture, however, yields a normal fluid which may or may not be under pressure.

In some cases the face is pale in others flushed or cyanosed. The physical signs are often very difficult to elicit, particularly in the early stages and in small babies. During the stages of congestion it is seldom possible to be certain of the slight lack of resonance over the affected area though on auscultation it is possible occasionally to find a definite area over which the stethoscope reveals feebler respirations than elsewhere. In the second stage the dullness becomes definite but is never flat as in pleural effusion. Bronchial breathing commences now to be heard over the affected area. Moist sounds are usually absent, though pleural sounds may sometimes be mistaken for râles and crepitations. Often however, it is impossible to detect any definite physical signs. This is particularly noticeable in apical pneumonias. It is always well to place the stethoscope in the axilla, as bronchial breathing may sometimes be heard there when all other areas are silent. Once resolution commences these silent areas of consolidation usually reveal themselves moist sounds of varying quality can be heard, and the physician is often puzzled at his previous failure to locate the lesion.

In those cases where the physical signs have been clear throughout, a considerable period elapses before the dullness completely disappears and the normal respiratory sounds

return Daily examination should be made carefully at this time if the temperature has not completely settled down or if it tends to rise again as this is the stage when empyema not uncommonly occurs

**Diagnosis** The diagnosis of pneumonia usually can be made from the general appearance and respiratory distress as described above Sometimes acute abdominal conditions or meningitis will be simulated and occasionally pyelitis may be mistaken for an early pneumonia In the cerebral cases a lumbar puncture will settle the difficulty in pyelitis the examination of the urine gives the diagnosis and only in the abdominal type can any real difficulty arise Here particularly in older children the problem may be very difficult When this state of affairs arises an x ray of the chest will always settle the matter

**Complications** Dry pleurisy is an almost constant complication of lobar pneumonia when the periphery of the lung is involved in the pneumonic process it may be associated with a slight serous exudate If the process stops here resolution will follow the normal course In severe cases however organisms and pus cells appear in the exudate which may thus become the beginning of an empyema or a pleuro pneumonia

### *Pleuro pneumonia*

This may be defined as the condition which occurs when a profound fibrinous pleurisy occurs associated with pneumonia Usually the entire pleural covering of the lung is affected as well as the parietal pleura The exudate forms a thick greenish yellow covering in which pockets of pus may sometimes be found The physical signs are often very difficult to elicit Pleural râles and crepitations are heard the percussion note is dull and sometimes flat the x ray gives an opaque shadow which may represent either fluid or thickened pleuræ and paracentesis may detect nothing or perhaps a few cubic centimetres of fibrinous exudate or pus may be drawn off Interlobar pleurisy and empyema not uncommonly occur associated with this condition and the mortality is high particularly in babies Resolution may be complete but not uncommonly dense fibrous adhesions form which bind down and distort the lung and may later lead to fibroid lung or bronchiectasis Otitis media is a common complication of

lobar pneumonia, and should always be kept in mind. As a bacteremia is commonly associated with the condition, septic complications elsewhere in the body are common, pneumococcal meningitis, parotitis, peritonitis, pericarditis, arthritis, malignant endocarditis, acute nephritis and hepatitis may supervene. The signs and symptoms of these conditions are found in all text books of medicine, and space does not permit of a description of them here.

**Treatment** The main principles of the treatment for lobular and lobar pneumonia are similar and hence both are discussed together below.

In infants every effort must be made to prevent the occurrence of the disease. Babies must be protected from upper respiratory infection as from the plague. The younger the baby the more important is this principle. Adults with colds and coughs must be kept away from babies. No adult suffering from any infection should be let into a room where a new born baby is being nursed.

Of recent years a great step forward has been made in the treatment of adult lobar pneumonia by the introduction of type specific *anti pneumococcus serum*. In Type I, and to a lesser extent Type II, this therapy has proved invaluable. It is necessary, however, to give the right specific type serum, and hence essential to collect sputum from the patient and type the infecting organism. Babies do not expectorate, hence typing is very difficult, and the reports in both lobar and lobular pneumonia of series of cases treated by pneumococcal serum have so far been disappointing. Therefore most writers do not advocate the use of serum in the treatment of pneumonia in babies. The author, however, has observed very beneficial effects in a number of cases treated by serum and believes that in time with further elaboration of serum therapy, this method of treatment will become much more successful. Vaccine therapy also has its advocates, who claim very satisfactory results from the use of pneumococcal vaccines given during the disease. Blood transfusion is also a measure of real value and may sometimes save a life. It is technically a specially difficult procedure in these cases, as the heart is often beginning to show right sided failure due to pulmonary congestion and more blood added to the circulation may embarrass it further. Small quantities of blood (5 cc per pound of body weight) if administered slowly enough

may be given with safety though the patient's condition must be watched carefully during the procedure. Sometimes it is wise to withdraw 10 c c of blood from the patient before commencing the transfusion.

The administration of oxygen is the most important therapeutic measure in the treatment of both lobular and lobar pneumonia. To be of use it must be employed in the right way. It is quite useless given out of a funnel held in front of the child's face. It must be given either by means of a nasal catheter or oxygen tent. The latter is a very valuable method in older children and one which should be employed in every hospital. In babies the nasal catheter is usually best as it is very difficult to nurse an infant in the usual oxygen tent. The catheter is covered with vaseline and passed up the nose and down into the naso-pharynx being held in position by a piece of adhesive tape fixed to the face. Enough oxygen must be given. Commonly we have observed in hospitals only a slow bubble being allowed to pass through the wash bottle with frequent intervals of complete cessation. Oxygen to be of full value must be given in sufficient quantities to prevent cyanosis and must be administered continuously while the patient shows respiratory distress. So as to avoid excoriation of the naso-pharynx the position of the end of the catheter should be altered from time to time and the catheter should be changed from one nostril to the other.

Stimulants except coramine are of very little value. The latter may be given in  $\frac{1}{4}$  c c doses six hourly even to small infants in cases of respiratory distress and often proves of the greatest value.

As sedatives when the child is restless and sleepless alcohol and luminal are of great value. Alcohol is best administered in fairly large doses occasionally rather than continually in small doses—e.g. brandy  $\mathfrak{M}$  xxi or sodium luminal gr  $\frac{1}{4}$  to a baby of three months old with advantage. The dosage in every case will be different. It is best to start with small doses and increase till the desired effect is produced.

Morphine and codeine should be employed with the greatest caution in babies. The present author never uses morphine for babies under a year of age as it sometimes leads to collapse.

In lobular pneumonia which has continued for a considerable time Still recommends —

Tinc Nucis Vom	℥	$\frac{1}{2}$
Syrup Scillæ	℥	$\frac{5}{8}$
Glycerin	℥	$\frac{5}{8}$
Aqua	ad	$\frac{3}{4}$ 1
℥i three hourly to a child of three months		

The author has found —

Tinc Nucis Vom	℥	$\frac{1}{2}$ —1
Tinc Stramonium	℥	$\frac{1}{2}$ — $\frac{1}{4}$
Pot Iodide	gr	$\frac{1}{4}$ — $\frac{1}{2}$
Syrup	℥	$\frac{1}{2}$
Aqua	ad	$\frac{3}{4}$ 1

℥i four hourly

to be of great value to babies with much secretion and some bronchial spasm, particularly in lobular pneumonia complicating whooping cough

The nursing of these patients is perhaps the factor of most therapeutic importance. When the child can have special day and night nurses his chances are very much improved. No type of nursing is more arduous, but none gives greater reward to perseverance and care.

In the infant the problem of feeding will be of paramount importance. Here each case must be treated on its own merits. In lobar pneumonia, while the fever is high the feed must be diluted, but enough fluid must be got into the child. Gastroenteritis often complicates the case and has to be treated separately. Restlessness associated with high fever is best treated by tepid sponging with warm water about 75° F. Localised pain may be relieved by antiphlogistic poultices or by an ice bag in older children, but poulticing as a general principle does more harm than good by overheating the patient. For the same reasons gamgee jackets are to be deprecated, and light woollen garments preferred. Inhalations in stuffy rooms and tents are useless, airy rooms or balconies in the open air (in warm weather) are the right places in which to nurse pneumonic patients.

### Interstitial Broncho-pneumonia

Interstitial broncho pneumonia is almost always secondary to some infection such as whooping cough, measles influenza, diphtheria or scarlet fever. It occurs most frequently under three years of age. Most of the above diseases, however, are

rare during the first four to six months of life and consequently the condition is not commonly met with till after six months of age. The mortality is extremely high and the outlook always grave. It tends to occur in epidemic form in institutions following some infection such as measles.

**Pathology** The essential difference between interstitial broncho pneumonia and disseminated lobular pneumonia is that in the interstitial form there is involvement of the supporting structure of the lung while in the latter only the mucous membrane and alveolar spaces are affected. In lobular pneumonia a bronchiole and its corresponding alveolar tree are affected. In interstitial pneumonia some of the affected alveoli belong to the same system as the affected bronchiole and others not but merely situated adjacent to it.

In interstitial pneumonia both lungs are usually affected small firm areas being found throughout at autopsy. On section pus will ooze from the cut ends of the bronchioles. On microscopic examination the affected bronchioles will be found full of exudate, areoli full of epithelial debris, leucocytes, red blood corpuscles and fibrin while the interstitial alveolar tissue is thickened and infiltrated with mononuclear cells. As the process proceeds new blood vessels invade the walls of the bronchioles and alveoli, new fibrous tissue is laid down and the alveolar spaces become obliterated. When the condition becomes chronic fibrosis of the lung occurs, the bronchi become dilated and distorted and eventually a condition of bronchiectasis supervenes. The latter is a gradual process and is outside the scope of this work which deals with the infant.

The commonest organisms associated with the condition are the hæmolytic streptococcus, Pfeiffer's bacillus and the staphylococcus though the infection is usually a mixed one and the pneumococcus, micrococcus catarrhalis and other organisms are often found as well.

**Signs, Symptoms and Diagnosis** It is often impossible to distinguish the condition clinically from lobular pneumonia as the signs and symptoms may be very much the same *e.g.* rapid respiration, swinging temperature, prostration, scattered areas of consolidation and varying moist sounds on auscultation. Certain points in the general disease picture may help however. In interstitial pneumonia the onset is usually gradual and follows some debilitating general infection such as measles or whooping cough. It runs a prolonged course, as relapses are

common, and it is frequently followed by empyema and chronic fibrosis of the lung

When whooping cough is complicated by interstitial pneumonia the onset is usually gradual during the second or third week of the disease appearing first as bronchitis the graver symptoms developing slowly In influenza, on the other hand the condition may develop synchronously with the primary infection or occur suddenly some days after it The course of the condition is almost always protracted, weeks or months passing while the child continues to show varying symptoms Gradually emaciation and anæmia occur, the child assuming slowly the marasmic facies If seen for the first time in this stage the condition may easily be mistaken for tuberculosis In these cases vomiting and diarrhoea usually occur, the appetite becoming worse as the condition proceeds Bed sores may form as the child becomes more and more cachectic On the other hand complete recovery may take place sometimes after one or two weeks of acute illness though this is a rare occurrence Occasionally after months of illness there is complete recovery but these long drawn out acute cases tend gradually to become chronic and lead eventually to fibroid lung and bronchiectasis as the child grows older

**Prognosis** The outlook is always serious and under one year few children recover When the condition occurs associated with some infectious fever in a home for infants, the mortality may be as high as 80 or 90 per cent

**Treatment** During the acute phase this does not differ essentially from that of lobular pneumonia As the condition progresses and the baby becomes increasingly emaciated and anæmic, no treatment is of more value than blood transfusion Expectorant drugs are useless, vaccines appear to do no good during the acute phase, and the treatment resolves itself into maintaining the child's strength by every possible means Good nursing is of the utmost importance for occasionally, if great care is taken the child will make a complete recovery after months of severe illness

The greatest care must be taken in all institutions and hospitals where children are kept in wards or nurseries Infectious fevers must be isolated, and if a case of measles occurs the other inmates of the ward should be given convalescent serum without delay (see p 153)

Finally the importance of regarding every case of respiratory

disease in children as infectious cannot be too strongly stressed. Hospital authorities are as yet uneducated in this matter. To them certain conditions are officially infectious diseases, measles, whooping cough, chicken pox, scarlatina, etc. The rules for dealing with these states are clear: immediate isolation of the patient and the ward in which the outbreak of the disease has occurred is ordered. If however the infection does not fall into one of these official categories, nothing is done.

All respiratory infections should be nursed in separate isolation cubicles. Hence all children's hospitals, more especially hospitals for infants, should adopt barrier nursing, and sufficient isolation should be provided in all homes for normal infants so that should an infection break out it can be prevented from spreading through the institution.

## CHAPTER VIA

W R F COLLIS

### EMPHYEMA

(Ætiology—Pathology—Symptoms Signs and Diagnosis—Complications—Prognosis—Treatment General—Paracentesis—Closed Drainage—Open Drainage—Summary of Treatment)

NINETY per cent of cases of empyema are associated with pneumonia usually lobar pneumonia and hence the commonest causal organism is the pneumococcus. During the neo natal period it is sometimes associated with umbilical or skin infection. Empyema may complicate measles whooping cough or scarlet fever. It may be associated with any suppurative process such as osteomyelitis appendicitis or pyæmia. Occasionally it follows the bursting of an abscess into the thoracic cavity from the peritoneum thoracic wall or mediastinum. Rarely in children and more rarely still in babies it is caused by an extension of a caseous tuberculous process. Hence although the pneumococcus is by far the commonest causal organism streptococci staphylococci Pfeiffer's bacillus tubercle bacilli or mixed infection may be found occasionally.

**Pathology** The empyemic process resembles that of simple pleurisy. At first the exudate is largely serous but if examined microscopically will be seen to contain micro organisms. It soon becomes purulent the type of pus depending on the causal organism. In streptococcal cases the pus is thin sometimes hæmorrhagic while in pneumococcal cases it is thick and greenish. The exudate pushes the layers of the pleura apart and covers the surface of the affected lung area often spreading beyond and covering the adjacent lobes as well. The exudate may be small and localised. Sometimes a small encysted collection is found between the lobes of the lung—an interlobar empyema. When the exudate is large the mediastinum is pushed away from the affected side and the pus unless evacuated may track through the thoracic wall and appear as a localised subcutaneous abscess—*empyema necessitatis*. Sometimes if the exudate is not large and the case is not diagnosed

the pus may become walled off and sterile, and remain thus for a long period. The two layers of the pleura tend to become closely bound together during the healing processes, and the lung may be dragged upon and distorted by dense layers of fibrous tissue.

**Symptoms, Signs and Diagnosis** Empyema following lobar pneumonia tends to present a very definite clinical syndrome. First the child runs the typical high temperature of lobar pneumonia, associated with the other signs and symptoms of the disease. The temperature then falls by crisis or lysis, and the patient appears much better for a day or so. Then the temperature again rises and becomes irregular, while the child

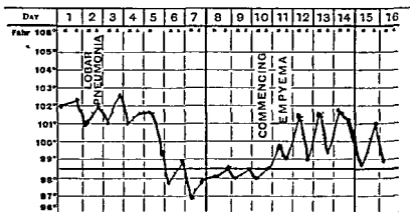


FIG. 20—Chart from case of lobar pneumonia showing commencing empyema

becomes increasingly distressed, tends to sweat, and wastes, showing more and more signs of anemia and prostration (see Chart). Careful examination reveals an area the commonest position being the left base over which dullness is found on percussion and where the breath sounds are absent. If the effusion increases rapidly, the heart becomes displaced towards the other side and attacks of syncope are apt to occur, particularly in infants. The diagnosis in these cases is not difficult. An x ray will reveal the presence of an effusion and paracentesis will demonstrate if the fluid is purulent. Such cases should never be missed, provided the doctor is aware that empyema is apt to complicate pneumonia, and is on the look out for the above clinical picture. Often, however, the doctor does not see the patient throughout the initial

illness and the baby is brought to him for wasting. The temperature may be normal and the child present a picture of emaciation lying limp and pale in the mother's arms. More than once we have had such cases sent to us labelled *marasmus* and on examination have found a large purulent effusion almost filling one side of the chest. Interlobar empyema may be impossible to diagnose without the aid of an x-ray.

In the diagnosis of effusion in the small baby nothing is so helpful as a trained sense of touch. If the middle finger of the right hand is tapped lightly over the chest wall a feeling of resistance will be encountered over the area of the effusion. This sign is much more easily elicited than the usual percussion sound which may be drowned in the baby's cries.

Skodaic resonance heard at the apex or over the anterior chest wall when the child is lying down is a sign of importance and when heard should always make the physician suspect fluid.

In early cases with large effusion the viscera will be displaced away from the lesion but in old standing cases the heart may be pulled towards the lesion by fibrosing adhesions.

A leucocytosis of 30 000–40 000 is usually present but as this is not an uncommon count in pneumonia as well it is seldom significant when taken by itself.

The only two conditions which are apt to be confused with empyema are unresolved pneumonia and generalised tuberculosis. The former particularly when of the pleuro pneumonia type may give almost the same signs and symptoms and only paracentesis will settle the matter.

**Complications.** *Suppurative pericarditis* is among the most important complications of empyema. It is very difficult to diagnose during life. It tends to occur most commonly during infancy. In the baby the signs are very hard to make out. The area of cardiac dullness will be increased the heart sounds will be faint and the skiagraph will show an enlarged pericardium. The baby is usually so acutely ill that these signs cannot be demonstrated. Still suggests that in a case of empyema where drainage has been established and the child does not improve but continues to run a hectic temperature and a fast pulse suppurative pericarditis should be suspected even when pericardial rubs cannot be heard.

*Pneumococcal meningitis* sometimes occurs associated with empyema particularly in young infants, suppurative arthritis

and acute peritonitis are rare complications. The latter is seldom discovered before it is too late for therapy, as the double diagnosis of empyema and peritonitis is very difficult to make. Lung abscess is a rare complication in infancy. If the cough suddenly becomes soft, abscess should be suspected and all attempts to produce expansion of the lung stopped.

**Prognosis.** The outlook depends upon the interplay of three factors: *the age of the child, the causal organism, and the treatment.* The disease has a mortality of nearly 50 per cent. under one year, after three years if treated early and satisfactorily the majority of patients make a complete recovery.

The disease is particularly fatal in infancy, because at this age the body has not acquired much resistance to the invasive properties of pyogenic organisms.

The hemolytic streptococcus is probably the most dangerous causal organism, being highly toxic as well as having a high invasive power, though less likely than the pneumococcus to cause pericarditis and meningitis.

The staphylococcal infections tend to have a better prognosis than streptococcal, while mixed infections have the worst outlook of all.

The prognosis in all cases will depend largely on early diagnosis, satisfactory drainage, and good nursing.

**Treatment.** The baby suffering from empyema should not be regarded as a surgical case. The infant should be kept in the medical ward and in charge of the physician, for he will require careful medical and dietetic treatment if his general condition is to be maintained. Too often these cases are abandoned to surgical departments where no doubt the drainage operation and dressings are carried out with all skill but where there is no real provision for infant dietetics.

In infants simple aspiration, repeated if necessary, will be successful in quite a high percentage of cases. When the pus is very thick or the exudate extensive it is usually necessary later to resort to some form of permanent drainage. In infants the closed method is the safest and should always be instituted first. It will succeed in a great many cases. Only when it has been tried and has failed to drain the cavity completely should open drainage be resorted to.

In older children although the above facts still hold good, they are not of such paramount importance, and open drainage may sometimes save time. In all big exudates, however, some

fluid should be drawn off by aspiration first, so as to allow the mediastinum to swing back gradually and thus avoid shock.

Infants suffering from empyema are almost always anæmic, and their general resistance usually is greatly lowered. Hence no therapeutic measure is more advantageous in these cases than a blood transfusion, which will make good immediately the deficiency of red blood corpuscles while at the same time providing the infant with leucocytes, immune bodies and food substances. Each individual case will need special arrangements regarding feeding and nursing, and the ultimate prognosis will often depend upon the success or failure of these measures.

Below is given briefly the technique of the various surgical measures used in the diagnosis and treatment of empyema in babies.

(1) **Paracentesis** The piercing of the pleura causes pain and shock, and hence it is always well to anesthetise the area to be pierced down to the pleura with procaine even in small babies. The needle used for exploratory puncture should be of wide bore and attached to a syringe with a good sucking power, as the pus is sometimes very thick and may not pass up the needle with ease. The position in which the baby is placed will depend upon the site of the exudate, and so, before paracentesis, a radiograph should always be taken if possible. If the pus is basal it is best to have the baby held in the upright position. In small babies the thoracic wall is very thin, and the needle must not be pushed too far as it may damage the lung or become plugged.

(2) **Closed Drainage** Various methods are employed in the different centres. A Rotanda 50 or 100 c c syringe is a very useful instrument for aspiration. The syringe has a special grip and a three way headpiece so that air replacement can be done if required. We have used this syringe with success in a number of cases in small babies, a couple of aspirations being sufficient to evacuate all the pus and complete recovery following. Gentle aspiration is most important as the Rotanda syringe is a powerful instrument and the infant's lung may be damaged if too great force is employed. The other methods of closed drainage consist in introducing a rubber tube between the ribs into the pleural cavity. Negative pressure is then applied to the tube and the pus slowly drawn off. Negative pressure can be obtained by various means. The end of the tube may simply be placed in a bottle containing fluid on the floor beside the bed or some form of suction apparatus applied, depending upon the rate of flow desired and the viscosity of the pus. This method saves the child continual dressings and impedes respiration very little. Elaborate methods have been invented. Hart has an

apparatus which supplies both negative pressure and continuous irrigation to the empyema cavity. Such methods are impracticable as a rule in the baby.

**Open Drainage** If closed drainage fails to evacuate all the pus open drainage must be resorted to. This can often be accomplished best in the small baby by an intercostal incision and the insertion of a rubber drain. In older children intercostal drainage seldom proves sufficient and rib resection is best resorted to as soon as open drainage is desired.

**Summary of Treatment for Empyema** (1) Aspirate under local anæsthesia—

(a) To obtain pus for examination

(b) To remove enough pus to correct any displacement of mediastinum

(2) Employ every means to restore child's general condition by diet, good nursing and transfusion if necessary.

In chronic cases or if the patient pass from the acute into the chronic state

(3) Endeavour to remove remaining pus by repeated aspiration. If this treatment fails

(4) Resort to continuous drainage—closed or open

## CHAPTER XX

W R F COLLIS

### TUBERCULOSIS IN INFANCY AND CHILDHOOD

#### (The Primary Complex)

(Importance of Subject—Modes of Infection—Epituberculosis—Diagnosis  
History Physical Examination Tuberculin Tests (Von Pirquet Moro Mantoux)  
Sedimentation Time  $\times$  Ray—Prognosis—Treatment Bovine Human )

TUBERCULOSIS in infancy and childhood is a subject of considerable complexity about which much controversy has taken place during the last two decades. The most divergent views in regard alike to its aetiology pathology and prognosis have been expressed often with considerable vehemence. Hence it is not surprising that the ordinary practitioner finds it very hard to get a clear knowledge of the subject. Gradually however the controversial points have been settled and a fairly definite agreement reached by specialists in all countries in regard to the main syndromes of the disease during the different age periods.

**Chances of Recovery** It used to be thought that the disease was almost always fatal during infancy and early childhood. Now we know that this is not so and if the case is diagnosed early enough and removed from contact before the child has had long or repeated infections that complete recovery is common.

**Importance of the Subject** In the National Children's Hospital in Dublin tuberculosis in all forms was responsible for one quarter of the total number of deaths in one year. In the Children's Hospital Birmingham one third of all deaths are due to tuberculosis or pneumonia the number caused by each disease being approximately the same\*.

It is generally agreed that the younger the child the graver the prognosis (see below) hence the importance of including a description of the disease in a work such as this with its emphasis on the infant.

\* Parsons L G *Lancet* May 20th 1934 1101

**Modes of Infection** If we except cutaneous manifestations of the disease there are three routes of entry for the germ into the body —

(a) By inhalation by way of the respiratory passages either by droplet infection or inhalation of infected dust

(b) By the ingestion of dust containing tubercle bacilli by children at the crawling age in tuberculous households

(c) By the consumption of infected milk

The actual percentages of the different types depend on the district from which the figures are taken Griffith has typed the strains (into human and bovine) from 188 cases of tuberculous meningitis taken from different parts of England and Scotland and has found the percentage of bovine (milk caused) cases to be as follows —

Age Periods	Percentage Bovine
0-1	15
1-2	40
2-3	46
3-4	23
4-5	30
5-6	30
6-7	60
7-14	14
15-24	10

No figures are as yet available from Ireland as far as children are concerned though an investigation is at present being undertaken by the author along similar lines to the above It is possible to say already however that the figures for Dublin city at least will show in all probability a smaller percentage of children dying from bovine tuberculosis Recently 360 sputa from cases of adult phthisis in an Irish sanatorium were typed all were found to be the human variety

The following diagram illustrates the *modus operandi* of tuberculous infection in its different forms —

Fig 21 diagram 1 represents diagrammatically how infection of the human type affects the child when first attacked A droplet containing a number of tubercle bacilli is inhaled by the child and the germs are lodged in the parenchyma of the lung A period from two to eight weeks now elapses before any clinical signs or symptoms appear At the end of this incubation period certain symptoms of the disease will appear, depending upon the severity of the dose that the child has received and

his state of health at the time. Anorexia, loss of weight, elevation of temperature (this is very irregular in extent and duration) and pleural pain occur in varying degrees and are associated occasionally with such paratuberculous conditions as phlyctenular conjunctivitis, erythema nodosum and early acute tuberculous cervical adenitis.\* Clinical examination of the chest at this time more often than not reveals nothing; occasionally fine crepitations can be made out or a unilateral

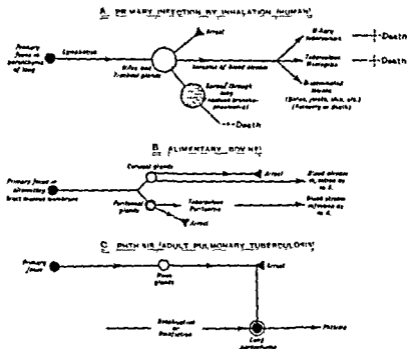


FIG. 21.—Diagrammatic representation of modes of tuberculous infection

bronchitis found, but the clinical signs are seldom sufficient by themselves to warrant a diagnosis. At the same time as these symptoms begin to manifest themselves the tuberculin reaction becomes positive, the sedimentation time of the erythrocytes shows an increase, and radiographs of the lung not uncommonly reveal what has been called "the primary complex." This consists of a pneumonia-like shadow in the lung tissue around the primary focus together with a variable swelling of the hilus glands.

\* Early acute tuberculous swelling of the cervical glands must be distinguished from late caseous cervical adenitis

The subsequent course of the disease now varies depending upon a number of factors (see Prognosis) it may either be arrested completely the primary focus being absorbed fibrosed or calcified and the same happening to the hilus glands or the disease may spread either through the lung itself becoming caseous broncho pneumonia or by way of the blood stream. If the infection of the blood is continuous the condition of acute miliary tuberculosis is produced and is followed by death (see Plate IV). If the blood stream infection is mild various metastases may occur which give rise to exudative lesions. If the lung is reinfectd through the blood stream temporarily a condition somewhat resembling miliary tuberculosis in its x ray appearances occurs but differs from the latter in that it is not necessarily fatal. If a metastasis lodges in the central nervous system and ulcerates through the meninges the condition of tuberculous meningitis supervenes.

Fig 21 B represents what happens in the alimentary type of the disease and is self explanatory when the sequence of events in Fig 21 A has been studied.

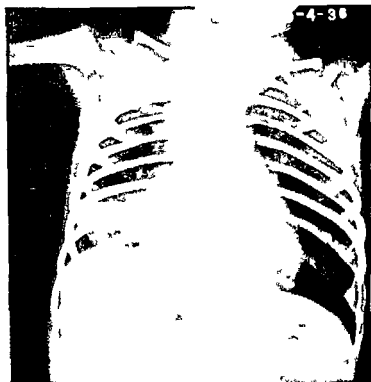
Fig 21 C shows how adult pulmonary tuberculosis or phthisis occurs. This takes place in later life when reinfection or reactivation of an old focus occurs in the case of a person who has been infected during childhood but where the primary infection had been arrested completely and the focus either absorbed or surrounded by fibrous tissue.

### Epituberculosis

We have already mentioned that the primary complex may show certain radiological appearances. In certain cases very large areas of the lung are seen to be opaque occasionally one or more lobes appearing completely consolidated. Study of the shadows and correlation with the clinical symptoms have shown that the condition is often benign that the child may be little if at all distressed run no temperature and take his food normally and that the whole state of affairs x ray appearances and clinical symptoms may clear up completely and leave no morbid sequela.

The term epituberculosis got into the literature some time ago to describe this condition and has remained ever since as a great source of controversy. Very few cases have ever come to autopsy and hence there is no clear foundation on which to base the pathology.

PLATE X



Showing interlobar effusion at right infero fissure also opacity at right apex. Child aged twenty months

The main theories to account for the x-ray appearances and clinical syndromes are as follows: (1) that the condition is caused by enlarged mediastinal glands pressing on a bronchus and causing collapse of the lung; (2) that it results from a chronic pneumonic condition in the lung of a non-tuberculous nature around a small tuberculous focus, the consolidation being brought about by a condition of hypersensitivity (or allergy); (3) that it may be a non-inflammatory reaction (*i.e.*, œdema); (4) that it may be a retrogressive tuberculous pneumonia. McDonogh has shown that a number of cases supposedly of epituberculous consolidation were, in fact, interlobar pleurisy, a thin layer of fluid lying obliquely and giving a shadow resembling consolidation (see Plate X).

The most recent work on the subject is by Oppenheimer, who, after much careful animal experimentation, comes to the conclusion that the lesion is essentially a tuberculous pneumonia clearing by resolution and organisation. She puts forward the hypothesis that the condition occurs when a caseous lymph node erodes a bronchus and discharges caseous tuberculous material, containing only a few live tubercle bacilli, into the lung of an already tuberculin hypersensitive individual. The few bacilli present and the tuberculo-protein then set up the reaction which causes these distinctive shadows to appear. This latter view is probably correct, though undoubtedly cases showing similar x-ray appearances have occurred due to pressure collapse or caseous pneumonia.

To pursue the matter further, however, in this work would only be to cause the student unnecessary confusion. The main fact to be grasped is that a benign condition with alarming x-ray appearances, but a good prognosis, not uncommonly occurs during primary tuberculous infection.

**Diagnosis.** It is not too much to say that the condition of primary tuberculosis is the most commonly missed diagnosis in the whole of medicine, for the reason that it not uncommonly occurs with few if any physical signs and that the patient may recover after only a short period of indefinite malaise; indeed recovery, particularly after the age of two years, is the general rule, as has been demonstrated by the tuberculin reaction which has shown that more than 50 per cent. of most adult populations are tuberculin positive, and hence must have passed through

the primary state at some period of their lives. As we have already seen however tuberculosis is one of the greatest causes of death in infancy and childhood. Therefore although the primary complex may be often insignificant its importance is very great as it may be followed by a fatal generalisation of the disease (acute miliary tuberculosis etc.). As we shall show generalisation is most apt to occur shortly after (within four months of) the primary infection hence the knowledge of how to diagnose the primary complex is of vital importance to any physician.

Below is given a summary of the methods of diagnosis employed by the author —

### Method of Diagnosis of Primary Tuberculosis

- (1) (a) *Family history*—question of contact in family
- (b) *Past history of patient and history of present illness*  
          with particular reference to symptoms such as  
          anorexia loss of weight slight temperature dry  
          cough erythema nodosum phlyctenular conjuncti-  
          vitis and pleural pain
- (2) *Physical examination*
- (3) *Tuberculin reaction*
- (4) *Sedimentation time*
- (5) *x Ray*
- (6) *Sputum examination (gastric lavage method)*

(1) History (a) A careful history must be obtained and any evidence of possible home contact carefully sifted. Often parents intentionally withhold knowledge of tuberculous contact for family reasons. The disease is erroneously considered hereditary in Ireland and carries a certain social stigma with it. Careful questioning combined with firmness and kindness in a room in which the physician and the parent are alone together will often elicit information which will be denied in the public clinic.

(b) Secondly a careful history of the case must be obtained. In infants anorexia loss of weight marasmus dry cough and temperature should be noted. In older children symptoms such as erythema nodosum conjunctivitis (phlyctenular) and pleural pain also aid the physician in making the correct diagnosis. In bovine tuberculosis affecting the peritoneal glands the first symptoms are usually vague pains often

mustal en for chronic appendicitis together with loss of weight and other similar symptoms as found associated with primary lung tuberculosis

(2) *Physical Examination* The reason why the diagnosis of the primary complex is so rarely made is because simple clinical physical examination is rarely sufficient by itself. The signs vary very greatly depending upon the degree of involvement of the lung and hilus glands. More commonly than not, nothing at all can be made out in the lung itself. Physical signs of enlarged hilus glands require a degree of faith unpossessed by the author who has never been sure of having found them in a single case in his experience.

When signs are present in the lung they vary from slight bronchitis to apparently complete consolidation of one lobe or even one whole lung. As a rule it is impossible by physical examination alone to be sure whether the process is of a pyogenic or tuberculous nature. A unilateral bronchitis affecting only a portion of one lung, pleurisy or consolidation without the other signs of lobar pneumonia will make the experienced pædiatrician suspect primary tuberculosis but it may be said generally that positive findings in the physical examination should never be regarded by themselves as sufficient data on which to base the diagnosis and negative findings should never rule out its possibility.

The early signs of tuberculous adenitis of the peritoneal glands are very vague. The abdomen may feel doughy but actual masses of glands are rarely palpable in the early stages. Once fluid is found in the peritoneum the disease has passed the glands and by the time the diagnosis has become obvious then the condition has advanced to that of tuberculous *peritonitis*.

(3) *The Tuberculin Test* Park has taught that the tuberculin reaction is the most important clinical test in pædiatrics. But in spite of this and the fact that the test has now been accepted by all modern pædiatricians as being of the greatest help in diagnosis not one in ten of general practitioners can be said to use it.

As we have said the tuberculin test becomes positive about two to eight weeks after the primary infection occurs and then remains positive in the vast majority of individuals for the rest of their lives though its intensity will vary considerably. Hence in adult life a positive test is of very little value. A negative test at any age however provided the patient is

not suffering from any acute infection (including generalised tuberculosis itself) at the time of the test is proof that the patient is *not* suffering from tuberculosis. In childhood the value of the test is much greater, as the proportion of positive reactions is very much smaller. The younger the child and the more secluded a life he has led the more valuable a positive reaction becomes. In any child under three years a positive reaction should make the physician suspect a recent tuberculous infection. Under two it is still more important and under one it indicates definitely infection in the last few months. Taken by itself the tuberculin reaction is only of absolute value when negative but when history symptoms and other laboratory tests suggest the disease a positive tuberculin reaction will often clinch the diagnosis. (See example at end of chapter.)

There are three tuberculin tests in common use —

- (a) The scarification or von Pirquet reaction
- (b) The injection or Moro reaction
- (c) The intradermal or Mantoux reaction

**Technique (a) von Pirquet Test** A drop of pure tuberculin is placed on the skin of the upper part of the right arm with the blunt end of a vaccinostyle. A scarification about 1 cm. in length is then made with the sharp end first on the skin some distance above the tuberculin and then through the tuberculin. The test may be regarded as positive when there is redness (with or without swelling) at the sides of the lower scarification and not at the upper — the test being read after forty-eight hours.

**(b) The Moro Test** Tuberculin ointment is employed in this test. An area about 4 sq. cm. is marked off on the skin of the chest and a small bead of ointment rubbed into it for thirty seconds. A control area may be mapped out and rubbed for thirty seconds with the finger if thought necessary. A positive reaction will be indicated by the appearance of one or more macules, papules or vesicles with a variable degree of erythema after forty-eight hours.

**(c) The Mantoux Test** This test consists of injecting 1 c.c. of a dilution of old tuberculin into the skin of the flexor surface of the forearm. A control may be done on the other forearm with glycerin veal broth (which is used in the preparation of tuberculin). The dilution used depends upon the circumstances. If the physician strongly suspects tuberculosis a dilution of  $1:1000$  should be used; otherwise a  $1:100$  solution should be the standard. If the latter is negative and the doctor still has reason to suspect tuberculosis a  $1:10$  or even  $1:5$  dilution may be employed. The reaction is read at the end of forty-eight hours. Usually a raised red area appears varying from the size of a shilling to that of half a crown. Some

times in strong reactions the centre becomes vesiculated and a wide area of erythema spreads around. In weak reactions there is no induration and sometimes there is only an area of erythema. In these latter cases it is best to repeat the test with a stronger dilution.

The Mantoux reaction is the most sensitive and accurate of the three methods but it requires the use of a hypodermic needle and occasionally severe reactions follow the injection of 1 c.c.  $\frac{1}{1000}$  dilution. Hence the Moro and to a lesser extent the von Pirquet tests may be preferred by some. In the author's experience the degree of sensitivity is usually high at the time when the primary infection is in its active phase and hence all three tests are satisfactory for the diagnosis of the primary complex. Once demonstrated to any student the technique is so simple and the advantages so obvious that no doctor's outfit should be without one of them. (The example given at the end of this section on diagnosis brings out the value of the test in practice.)

The question is often asked whether patients suffering from bovine tuberculosis react equally well to human tuberculin. In the author's experience the reaction is usually positive but less markedly so in these cases. This may mean that the bovine type of infection produces less hypersensitivity or that human tuberculin is not entirely interchangeable with bovine as has been thought by some.

(4) Sedimentation Time. This test has now come into general use as an indication of the activity of tuberculous disease. It is not specific and will also be high in any infective state particularly acute rheumatism. Its chief advantages are that it is often raised when the temperature and pulse are normal and that it has a wide range (0-70 mm. on our scale). We have used it as a routine on all our cases of primary tuberculosis for some years and have come to regard it as a very great help in gauging the activity of any given case.

**Technique.** The test consists in taking 4 c.c. blood by finger prick or venous puncture adding 1 c.c. sod. citrate 3.8 per cent mixing and running the citrated blood by capillary attraction into a small glass tube which is then set upright in plasticine. As the red blood corpuscles settle down in the tube they leave a column of clear plasma above. The length of this column of serum is measured in millimetres at the end of the first hour and the number noted. Below 10 mm. is considered normal. Severe cases may be as high as 60 mm.

There are numbers of different modifications used for the test in different centres, all are simple and can be learned at one demonstration and afterwards carried out by any student.

(5) *x*-Ray Skiagraphs of the lungs will show primary foci in the parenchyma and enlarged hilus and tracheal glands. The shadows may be very varied in size and degree and are sometimes difficult or impossible to differentiate from shadows caused by pneumococcal infection. A child should never be diagnosed on *x* ray appearances alone or very grave mistakes may be made and a child sent away to a sanatorium when it is not suffering from active tuberculosis at all. In fact no case should be diagnosed as tuberculous (excepting of course cases of miliary tuberculosis and meningitis) in which the tuberculin test has not been found positive.

The *x* ray appearances when taken together with the other findings are undoubtedly of the greatest help both in elucidating the complex ætiology of the disease and in following the course of any particular case. As we shall see when discussing the prognosis it is often astonishing how a large opaque shadow will disappear gradually and leave no sequelæ.

*x* Ray of the abdomen will reveal nothing in the early stages of tuberculous adenitis of the peritoneal glands. Not till healing and calcification occur will the glands cast a definite shadow on the skiagraph.

(6) *Sputum Examination* Direct sputum examination in small children is impossible as they always swallow what they cough up. However by washing out the stomach and centrifuging its contents it is possible to obtain their sputum which if treated with antiformin and injected into a guinea pig will cause tuberculosis in the animal if tubercle bacilli are present. Direct examination of the stomach contents under the microscope is not very satisfactory as other acid fast bacilli besides tubercle may be present and lead to confusion.

The lavage method is of no real practical value in the ordinary diagnosis of a case as six weeks must elapse before the animal shows the infection. It has proved of very great value in investigation on the subject however by demonstrating the ætiology of the primary complex. Some investigators have recovered tubercle bacilli from children with active primary tuberculosis in as high as 50 per cent of cases. The author in one investigation on cases of erythema nodosum obtained human bacilli in 25 per cent of his series.

**Example** The following example demonstrates the methods of diagnosis described above

A child aged three and a half years was admitted to hospital with tuberculous meningitis and died. The whole family were therefore sent for to see if either of the parents was the source of infection and if any of the other children had been infected. On examination the father was found to have a cavity in the right lung and a positive sputum. There were three other children in the family aged nine months, two years and six years.

*The nine months baby* was still breast fed, was over 14 lb in weight and appeared healthy in every way. The tuberculin test was negative. He was kept under observation for six weeks and then discharged as non infected.

*The baby of two years* gave a history of being off his food for the last month, sweating at night, crying more than usual and being out of sorts generally. The physical examination revealed a patch of bronchitis in the right lower lobe but nothing else. The temperature was normal. The tuberculin reaction was strongly positive. The sedimentation time was 25 (normal 10). The x ray of the chest showed an area of infiltration in the middle of the right lower lobe and considerable enlargement of the right hilus glands with some lung infiltration around them. The diagnosis was therefore made of an active primary complex and the child admitted to hospital where he was rested, given good food and later sent to a convalescent home when the sedimentation time had reached normal. He made an uninterrupted recovery, all signs having disappeared in six months.

*The girl aged six years* She had no suggestive history and her physical examination revealed nothing abnormal. Her tuberculin reaction was positive. Her sedimentation time was 5. x Ray of the lung showed some increase of shadow at the hilus but no active foci in the lung. She was diagnosed as a child who had passed through the primary complex and was now inactive. As the father had been removed at once for treatment to a sanatorium she was kept at home though directed to attend the clinic every few months for observation.

**Prognosis** In the past the prognosis of tuberculosis in childhood was considered almost hopeless. Still it is not uncommon to meet such statements as "every child under one year contracting any form of tuberculosis always dies" (Frew). In point of fact of course the vast majority of children pass through the primary complex without further spread of the disease and make a complete recovery. The error has arisen in two ways. Firstly in the past minor degrees of the primary infection were never diagnosed at all, physicians only recognised the graver complications of the disease (such as menin-

gitis etc.) Secondly such careful observers as Blacklock got a somewhat wrong impression by studying the subject simply from morbid specimens in the post mortem room. In 1800 consecutive autopsies he found tuberculous lesions in 283 and of these 90 per cent had died from tuberculosis. Hence he assumed that if tuberculous infection occurs in childhood it is usually fatal. The fallacy is that he was dealing with a very special group the most diseased group it is possible to find: the children from an industrial town actually dying of disease.

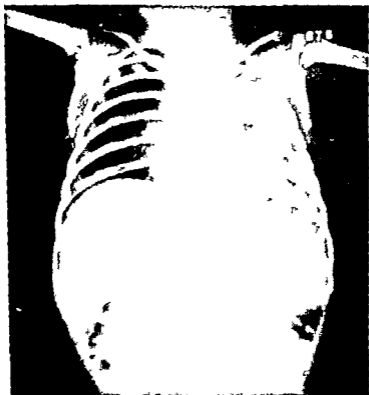
Some figures which we have collected recently in Dublin show how wrong such a view is. In my clinic for children suffering from primary tuberculosis some 120 have been kept under observation for the last three and a half years. Of these only one has died from tuberculosis. The ages of the children vary between one year and fourteen years the greatest frequency being at about seven years. Dr Price in a similar clinic at the Infants' Hospital St. Ultan's has observed for a similar period forty-four babies under five years of age with primary tuberculosis involving the hilus glands. In thirty-three the condition completely resolved in ten it is healing still and only in one has the focus broken down and led to death. During the same period thirty-five cases of tuberculosis which had not come for treatment till the primary focus had generalised, came to autopsy in St. Ultan's Hospital thirty-four of whom were under two years of age.

It would appear then that although tuberculosis in childhood has as high a mortality as any other disease yet many patients recover completely without sequelæ of any kind. What then are the factors which influence the prognosis? In this connection Borrell's work on African troops during the war is of importance.

These troops were brought over to Europe for the first time during the war previously they had lived in the comparatively tuberculosis free environment of North Africa and some 90 per cent were found to be negative to the Mantoux tuberculin test.

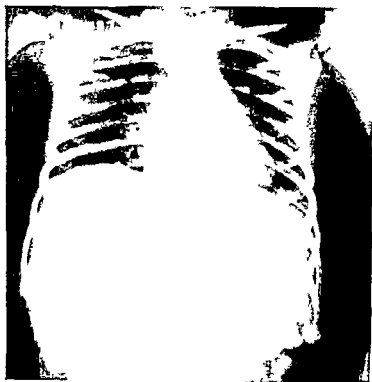
Borrell was able to show that these men reacted to their first tuberculous infection in a very similar way to the European child. After exposure to infection a period of three to four months was observed during which they became positive to tuberculin test showed loss of weight slight fever alimentary

## INITIAL



Skin grafts showed no primary complex in a child of eighteen months—  
nephritis in the left leg. Same was sixteen in the later of wing elongation  
of joints in time. The child made a complete recovery.

# PLATE VII.



disturbances, and increased hilar shadows in the x rays of the lungs

At the end of this period they either recovered completely, or the disease progressed and became generalised with fatal results.

Borrell was also able to demonstrate that the prognosis depended very largely on the treatment they received during this initial period. If they were taken off heavy duty and given rest, fresh air and a full diet, the majority recovered, while if they were left at the front or kept on strenuous fatigue duty at the base, the disease tended to get the upper hand and to generalise.

His work demonstrates that the prognosis in the primary complex depends very much upon the treatment which the patient receives. If the condition is not diagnosed and he is allowed to carry on his normal activities, and perhaps contract some intercurrent infection, such as whooping cough, the disease tends to generalise. If, on the other hand he is diagnosed early and rested, given a generous diet and protected from intercurrent infection, he tends to get quite well. Wallgren has shown that the dangerous period in which generalisation tends to occur is most commonly shortly after the primary infection, *e g*, in from six weeks to four months.

Another factor of importance in the prognosis is the age of the patient. Children under two years are much more likely to pass on from the primary complex without resolution to miliary tuberculosis, tuberculous meningitis and caseous broncho pneumonia than older children. It appears that their defensive tissue reaction to the disease has not been fully developed at this age. Blacklock has shown that the younger the child the less the tendency to fibrosis and calcification. He found that it is not till after two years of age that fibrosis is the rule, under two years he found evidence of calcification in only 14 per cent of his cases, while it was present in 36.1 per cent in cases over two years.

Another factor which undoubtedly increases the mortality of the disease among infants is the number of tubercle bacilli inhaled. The source in infancy is usually the mother or some near relative. The infant in being nursed is exposed to much droplet infection on repeated occasions. He lies in his cot unable to move about, and is seldom diagnosed before he is heavily infected. The same principles hold good in tuberculosis affecting the alimentary system. If diagnosed early

and taken off their feet and removed from contact most patients make a satisfactory recovery though again under two years the prognosis is very much graver (See Plates VI VII XIA and XIII)

**Treatment Milk, Bovine Infection** It is calculated that 7 per cent of the milk supply in Great Britain contains tubercle bacilli and that the treatment of bovine tuberculosis costs £500 000 a year In Ireland we have no comparable figures but if we are to judge by the number of children with bone joint and glandular tuberculosis as some gauge of bovine infection the figure must be considerable Such a state of affairs is completely inexcusable It means simply that we are allowing many of our children to be crippled for life and condemning many more to death every year when the remedy is in our hands The problem merely needs a little clear thinking Bovine tuberculosis infection could be abolished if the following methods were put into action —

First make the sale of tuberculous milk illegal Then every effort should be made to obtain tuberculous free herds and to encourage the production of F T Grade A milk For the smaller farmer and the poor man this can only be done with State aid and the process must be slow in any case In the meantime it is only necessary to insist that all milk other than F T shall be pasteurised in up to date State controlled pasteurising centres Where these measures have been adopted as in the U S A Canada and different parts of Europe bovine tuberculosis has become a medical curiosity

In democratic countries however politicians only act when public opinion forces them to do so otherwise they tend to evade the issue for fear of losing votes Hence it is necessary for the medical profession and public health services to educate public opinion up to the point where they will demand a pure milk supply Much has been done in Dublin recently but until it is illegal to sell milk containing live tubercle bacilli we must teach every mother to boil cow s milk unless it is Grade A F T

It is most important that the milk shall not only be boiled during infancy but right through childhood for if Griffith s table on p 218 is referred to again it will be seen that whereas only 15 per cent of children dying under one year of tuberculosis are infected with the bovine bacilli 46 per cent are so infected between two and three years

It has been suggested that the ingestion of tubercle bacilli

# PLATE VII

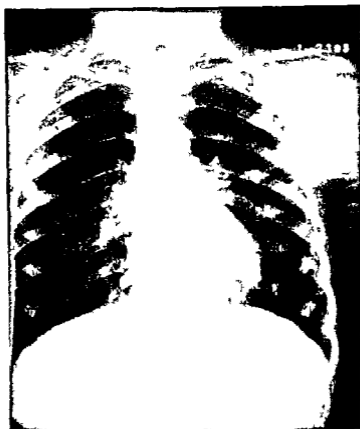


# PLATE VIII



Showing triangular shadow on right lung and gross enlargement of hilar glands. Age 1 1/2 years. The child had erythema nodosum at this time and tubercle bacilli were obtained in the gastric lavage. The mother had phthisis. Same case one year later showing clearing of the lung. The child made a complete recovery.

# PLATE XIII



## HEALING GRONF

Skiogram showing lung with numerous small nodules in the parenchyma (nodules of infection) and much consolidation in the right lung is in a child three to four years after primary infection. The child is at present in good health.

of immunity If it is to be used the most stringent precautions are necessary in its preparation for a disaster has happened where cultures of virulent tubercle bacilli got mixed up with the avirulent cultures and many deaths resulted Vaccination with heat killed tubercle bacilli also has its advocates (E Langer) and recent animal work by Walter Pargel shows that heat-killed vaccination if sufficiently thorough protects animals even against virulent infections

**Cure** The treatment of the child during the primary infection consists in (1) immediate elimination of the source of infection so that the child shall not receive further infection In the case of bovine infection this is simple, but when one of the relatives has active tuberculosis it may take time, and in this case the baby (or child) is best removed to hospital at once (2) Complete rest is essential while the disease is active Here the sedimentation time is of the greatest assistance to the physician As long as it indicates activity the child must be kept in bed In all our cases it is carried out weekly and by its help we are able to regulate the amount of rest necessary In some cases only a few weeks of absolute rest are necessary, in others months are required The rest procured by taking a child off his feet is considerable and allows him to use the reserve of strength thus gained to fight the disease

Small infants are in as complete a state of bodily rest already as can be obtained for them They are using all their available strength for growth and development, and hence their treatment is difficult and often unsuccessful Also children between the ages of two and three are very hard to keep in bed, particularly in the home In hospital with the routine of the ward this is not so difficult (3) A carefully regulated and balanced diet is also of great importance We consider that the reason that many babies of the poorer classes do much better in hospital than at home is due to the fact that while in hospital their diet is regulated scientifically and contains ample vitamins as well as sufficient substances of caloric value and calcium and phosphorus (4) Infants and older children should be nursed as far as possible in the open air, but not exposed to the sun (5) They must be guarded against all forms of intercurrent infection For this reason the medical wards of a children's hospital is not the best place for them Open air convalescent homes are the ideal for these patients In such surroundings recovery will be the rule.

*Tuberculous Peritonitis and Surgical Tuberculosis*

The general hygiene and dietetic principles in the treatment of tuberculous peritonitis gland, bone and joint tuberculosis, differs in no way from that of primary pulmonary tuberculosis, except that treatment tends to be more protracted in certain cases and prolonged open air treatment is often necessary. In the case of tuberculous peritonitis certain general measures are often recommended (a) mercurialunction, (b) tuberculin (c) surgical drainage. The former has still many adherents but tuberculin therapy and surgical drainage are almost always contra indicated in the author's opinion, during the first two years of life.

If diarrhoea is a marked symptom kaolin bismuth and aromatic chalk may be used and combined cautiously in severe cases, with small doses of opium.

The surgical treatment of gland bone and joint tuberculosis does not concern us here.

*Generalised Tuberculosis*

The common forms of generalised tuberculosis—miliary tuberculosis caseous broncho pneumonia and tuberculous meningitis—and the localised forms such as caseous cervical adenitis, dactylitis tuberculosis of the kidney etc are fully described in all general text books and a description of their pathology, symptom complex, etc, is unnecessary here. The differential diagnosis of tuberculous meningitis is described on p 173.

## CHAPTER XXI

W R F COLLIS

### CONGENITAL SYPHILIS

(Pathology—Symptoms—Diagnosis—Prognosis—Treatment)

If either a mother or father is syphilitic at the time of conception it follows usually that the child will be infected unless the mother receives treatment during her pregnancy.

It used to be held that a syphilitic father could infect his child without infecting the mother as it had been observed that mothers bearing syphilitic babies often showed no signs of the disease themselves. Recent serological studies taken together with such observations as Colles's Law (a syphilitic baby can suckle its mother's breast without danger of infecting her but *not* that of a wet nurse) go to prove that although the woman may show *no* signs of syphilis at the time she is suffering from it nevertheless in a latent form.

The stage of pregnancy when the foetus is infected is important the earlier it occurs the worse the prognosis. If infected at or shortly after impregnation of the ovum early death of the foetus and abortion usually occur while if the foetus is infected shortly before or during birth no symptoms of the disease will appear till two to twelve weeks after birth.

**Pathology.** It is difficult to classify the lesions of early congenital syphilis some being secondary and some early tertiary. In late congenital syphilis the lesions are tertiary.

**The Liver.** This is probably attacked more often than any other organ due to its place in the foetal circulation (see p 170). The congenital syphilitic lesion in the liver is characteristic consisting of a pericellular interstitial cirrhosis. Later gummata may develop. The capsule is thickened and may form adhesions to surrounding structures. Microscopically a great increase in fibrous tissue is seen around the individual cells. In certain cases milary syphilomata are found in the liver in early infancy. The liver may be much enlarged at birth and found to be swarming with treponema.

*The Bones* The mechanism whereby the organism effects bony change is not altogether understood. It is considered by some to be a nutritional disturbance at the point of ossification and resembles the lesion seen in rickets the latter is due however to a disturbance in the calcium and phosphorus metabolism whereas syphilis produces its effect by disturbing the blood supply to the bone and cartilage. Microscopically an irregular yellow line is seen at the epiphyses due to an increase in the zone of early calcification. Submetaphyseal rarefaction is a characteristic feature of the disease and one of the diagnostic *x* ray signs. It is particularly well seen in the tibia though local areas of rarefaction may occur anywhere in the shafts of the bones giving them a *moth-eaten* appearance in the skiagraph. Osteomyelitis and osteitis are found in shafts of the bones which tend to be replaced by connective tissue and fresh bone laid down under the periosteum. Syphilitic periostitis is characterised by its patchy distribution. Epiphyseal separation is also generally described as a common occurrence. Actually the term is a misnomer as the break occurs through the weakened trabeculae at the end of the shaft. The separated epiphyseal end of the bone may be dislocated backwards forwards sideways or become impacted into the shaft (see *x* ray appearances p. 117).

*The Spleen* The spleen is usually much enlarged in new born children suffering from syphilis but shows no characteristic microscopic changes.

*The Lungs* In still born syphilitic infants or those dying shortly after birth a condition called white pneumonia is sometimes seen. The lungs are hard and contain little air the alveoli being full of leucocytes. The interstitial tissue is increased and the lungs are nearly white in colour. Occasionally small scattered gummata are found.

*The Blood* In the neo natal period a severe anemia is occasionally seen in syphilitic infants.

*The Alimentary System* Diffuse fibrosis of the submucosa of the small and large intestines is often found in infants dying of severe congenital syphilis during the first few weeks of life. Miliary gummata are also sometimes found in the intestinal walls.

*Sense Organs* Syphilitic otitis is seen occasionally as a complication of the rhinitis and pharyngitis. Iritis is an early sign and may be found at birth or may occur during the fourth

or fifth month. Choroiditis is common in early syphilis but is seldom associated with optic atrophy. Interstitial keratitis does not occur in early congenital syphilis but is one of the most characteristic features of the late stage.

**The Kidneys.** The new born syphilitic infant may show numerous pathological changes in the kidneys. Their growth and development may be retarded. Areas of perivascular round-celled infiltration may occur and occasionally interstitial fibrosis is found. These patients not uncommonly develop acute nephritis. The appearance of the glomeruli is the same as that seen in acute nephritis unassociated with syphilis; hence some authorities think that the nephritis occurring in such cases is due merely to some secondary bacterial cause.

**The Nervous System.** Early congenital syphilis may affect the brain or the blood vessels which supply it. Encephalitis and chronic meningitis are found. Occasionally adhesions obliterate the foramen of Magendie and hydrocephalus results. Miliary gummata may be found in the basal ganglia. Endarteritis is commonly seen in these cases with perivascular round-celled infiltration and interstitial fibrosis.

**The Skin.** See p. 324.

**Symptoms.** Infants born alive with the florid symptoms of syphilis such as bullæ rarely survive more than a few days even when treatment is instituted immediately. Generally frank symptoms of the disease appear about the third to sixth week.

The first symptoms are usually *wasting rhinitis* (snuffles) and *skin rashes*. Wasting may be the only symptom. The baby at first fails to gain weight then loses gradually becoming emaciated and wrinkled. The hair falls out. Anæmia appears and the skin takes on a *café au lait* appearance. Snuffles (see p. 423 Ear, Nose and Throat) usually begin about the sixth week. The nasal mucous membrane is excoriated and there is a discharge from the nose. A hoarse cry is often associated with the snuffles and denotes laryngitis. The skin rashes appear about the same time and are very variable in appearance. Most commonly they are erythematous but they may be papular, vesicular or bullous. The commonest skin eruption occurs on the buttocks and down the backs of the thighs and on the face. It is erythematous and resembles the common napkin rash. (For detailed description and differential diagnosis of these eruptions see p. 324.)

*Fissures* and *mucous patches* may appear on the lips and around the anus. *Condylomata* are found in regions of moisture and friction such as around the anus and vulva. They seldom occur before the latter half of the first year.

*Epiphysitis*. Sometimes a condition of pseudo-paralysis due to syphilitic epiphysitis occurs in a baby a few weeks old. One arm is usually affected, showing swellings about the joints. The arm may be held in the position adopted in Erb's paralysis. The baby appears unable to move the limb and resents it being touched. The temperature may be raised (e.g., 100° F.). The diagnosis may be difficult if the condition is unassociated with other signs of syphilis. Scurvy, osteomyelitis, acute epiphysitis, poliomyelitis and Erb's paralysis must all be considered. A history of sudden onset should rule out birth trauma and careful questioning of the mother may reveal a syphilitic family history. If separation has taken place, crepitus can be elicited. Diagnosis may be very difficult and as a rule cannot be made for certain without x-ray and serological corroboration.



FIG. 22 — Baby with congenital syphilis  
—hands showing scaling

*Syphilitic dactylitis* is a common symptom; it usually occurs in the proximal phalanges, appearing as a spindle-shaped painless swelling. It occurs most commonly in the middle third of the first year, and is usually associated with other bone manifestations of the disease. The diagnosis from tuberculous dactylitis may be difficult, but will be confirmed by x-ray and serological examination.

Syphilitic babies tend to be premature and weakly and, unless treated early, growth will be retarded. Rickets is very often found associated with congenital syphilis. For this reason *craniotabes* occurring in syphilitic babies during the first

two months of life gave rise to the belief in the past that congenital syphilis was a cause of the condition. The present view is that craniotabes is solely a rachitic phenomenon and when seen in syphilitic children indicates that the two diseases are occurring simultaneously.

The nails show certain peculiarities in congenital syphilis. Exfoliation and destruction may occur or the nail may show a high arched dorsum as if it had been pinched up by a pair of forceps.

Scaling of the soles of the feet and palms of the hands is a very characteristic sign of the disease. (See Figs. 22 and 23.)



FIG. 23.—Baby with congenital syphilis—feet showing scaling.

The lymph glands often show general enlargement. Hutchinson laid stress upon the importance in diagnosis of the epitrochlear glands. He stated that if these lymph nodes are found enlarged without other apparent cause in a baby, congenital syphilis should always be considered as a possible diagnosis.

The central nervous system is not uncommonly involved in congenital syphilis (about one third of the cases

with a positive Wassermann reaction in the blood show a reaction in the cerebrospinal fluid though this does not necessarily mean clinical neurological findings). Occasionally hydrocephalus due to syphilitic meningitis is seen. Sometimes head retraction and even opisthotonos occur and convulsions are not uncommon.

If the fundi are examined choroiditis without optic atrophy may be seen.

The spleen is usually enlarged and palpable.

*The liver* may be much enlarged even at birth. *Jaundice* is often an early symptom in children born with frank symptoms of the disease, or may be the first symptom to occur, or may appear later in association with other general symptoms between the fourth and eighth week. It is always a grave sign.

*The late symptoms of congenital syphilis, occurring from the third to tenth year or later* are outside the scope of this volume. They are altogether tertiary phenomena akin to those seen in acquired syphilis. They occur in children who have had no symptoms at or shortly after birth, or in those whose early symptoms were so mild as to pass undiagnosed. One point here is worth stressing—the characteristic congenital syphilitic Hutchinsonian teeth appear with the second dentition, and are never seen in the first.

**Diagnosis** The problem of the diagnosis and treatment of congenital syphilis may be divided into that (a) of the neo natal period, and (b) later.

As we have seen, babies born with frank symptoms of congenital syphilis are usually still born or die shortly afterwards, and syphilitic babies born without such symptoms seldom develop them before the fourth week. The diagnosis and treatment of this latter group presents a very serious problem to the doctor. Serological tests in the baby at this age are uncertain. (A syphilitic infant may not have developed antibodies to the disease and hence may give a negative Wassermann reaction or a positive Wassermann reaction may be found temporarily in a new born baby who has acquired the antibody from a syphilitic mother, and later the infant may become negative never showing any clinical signs of the disease.) If anti syphilitic treatment is started before diagnosis is definitely made the doctor may be left in a dilemma. On the one hand it is clearly inadvisable to give a prolonged course of anti syphilitic treatment to a baby who is only suspected of the disease, but, on the other, it is equally inadvisable to discontinue treatment too early and possibly leave the baby to develop signs of the disease later. Again, if no treatment is given during the neo natal period in a suspicious case, the child may go down hill, lose weight and become marasmic before the frank symptoms of the disease appear. Much valuable time may be lost and therapy begun too late to cure the baby. In our experience the best routine is to regard every baby born of a mother with a positive Wassermann (particularly if she has

had previous still births or abortions) as potentially syphilitic and to treat it accordingly. We have in this way we believe saved a number of babies who would have died almost certainly if treatment had not been commenced at once e.g. —

Recently we had a mother admitted to hospital who had had ten still births or abortions. She was forty years of age and near term once more. Her Wassermann was +4. The baby was four weeks premature but 7 lb in weight and apparently in good condition. Anti syphilitic treatment was commenced at once. But in spite of this the baby began to lose weight rapidly, became anæmic and by the tenth day had developed the typical *café au lait* appearance. Treatment was intensified, a transfusion was performed and the disease got under by the sixth week when the child commenced to gain weight and from then on made an uninterrupted recovery.

Diagnosis after the neo natal period is nearly always possible though often difficult. First we have the signs and symptoms —wasting rashes scaling of skin on hands and feet snuffles epiphysitis choroiditis etc. The differential diagnosis of these has already been discussed when they were separately described above. Sometimes if only one symptom or sign is present diagnosis may be impossible as many of the symptoms such as the erythematous rash or snuffles are more commonly caused by other conditions than syphilis. Under these circumstances the diagnosis will need corroboration from serological tests and by x rays of the bones. We have already mentioned the difficulty of accepting the evidence of the Wassermann reaction in the infant at birth. As a rule however by the sixth week it will be positive in the presence of syphilis. Sometimes though very rarely a woman with a negative Wassermann will give birth to a syphilitic baby. A single Wassermann reading should never be regarded as final evidence of the presence or absence of syphilis. In doubtful cases the Kahn flocculation test should be used. It often proves of value under such circumstances and x rays of the bones may give conclusive evidence of the disease. See p. 117 for Differential x Ray Diagnosis from Rickets and Scurvy.

**Prognosis.** Congenital syphilis causes a heavy ante natal mortality due to abortion and still birth. Also children born with symptoms of syphilis have very little chance of recovery. Children developing symptoms after the neo natal period have a good chance of cure if treatment is prompt and properly carried out though after effects may be found later associated

0.5 c.c. for another four to five weeks. Then give a month off treatment then another monthly course of weekly doses of 0.5 c.c. and so on up to the end of the sixth month. If the Wassermann reaction is still positive bismuth treatment is continued or arsenic substituted. If the Wassermann is negative it is wise to give one more course of bismuth during the second six months. If all is satisfactory the child can be discharged at the end of the first year.

If the injections are commenced during the first week and if the baby is going down hill it is sometimes wise to give two injections a week though the total amount of bismuth should be the same.

The doctor must be constantly on the look out for toxic symptoms due to the cumulative effect of the drug *e.g.* albumen in the urine of babies, blue line in the gums and stomatitis in older children. It is unwise to combine bismuth therapy with mercury or arsenic.

If a nasal discharge excoriates the anterior nares or if fissures appear round the anus a mercurial ointment should be applied to the affected areas.

In severe cases with marked symptoms and particularly when the cerebrospinal fluid gives a positive Wassermann reaction, more vigorous treatment becomes advisable and one of the arsenical preparations may be used. Sulphostab may be given intramuscularly at weekly intervals in the following doses: 0.05 c.c., 0.1 c.c., 0.15 c.c., 0.2 c.c., 0.25 c.c. etc. for eight injections. This treatment may be combined with pill protiod gr.  $\frac{1}{4}$  twice a day to a child under six months and gr.  $\frac{1}{4}$  after that age. If diarrhoea occurs the dose should be reduced.

Recently preparations of arsenic for oral administration have been used with considerable success (stovarsol or ovarson). We have no personal experience of their use in infants. In older babies and children they appear a most valuable method of therapy.

Some people are hypersensitive to arsenic but this condition is seldom seen in the baby. Should it occur the antidote is sodium thiosulphate given intravenously.

Most important of all in the treatment of early congenital syphilis is a properly organised clinic and social service. Without this it is impossible to follow up the cases discovered in the maternity hospitals, children's hospitals and child welfare centres. The clinic must be provided with laboratory facilities.

## CHAPTER XXII

W R F COLLIS

### UROGENITAL CONDITIONS

(Urine in Neo natal Period—Examination of Urine—Hæmaturia—Hæmoglobinuria—Bladder Control—Malformations Congenital Cystic Kidney Hydronephrosis (Intravesical Supravesical) Extopic Vesicæ Patent Urachus—Infections Pyelitis (Pathology Symptoms Diagnosis Treatment)—Perinephric Abscess Urinary Calculi—Genital Organs)

THE length of time from birth at which the infant first passes urine is variable. Occasionally no urine is passed for as long as forty hours. In these cases sometimes the bladder is found distended at others almost empty. Hence it would appear that the condition may be due either to failure of the neuro muscular reflex or of the kidneys to secrete urine.

The quantity of urine also varies greatly. The following table given by Holt represents the average figures for healthy babies —

1st day	amount of urine	0- 60 cc
At end of 1st week		100-200 cc
1st month		150-400 cc
6th month		250-500 cc
1st year		300-600 cc

During the neo natal period the urine shows a number of peculiarities. It is often highly coloured due to urates and uric acid which are in excess of the normal at this time. Not uncommonly albumin is found during the first week. This may be due either to a large number of epithelium cells or to a definite albuminuria. The latter condition we have constantly found in the babies of toxæmic mothers (particularly eclamptics) and is due probably to damage of the infant's kidney by the maternal toxin. It passes off usually after a few days without leaving any permanent after effects. Slight albuminuria is said to occur at times throughout infancy without ill effect. After the neo natal period the urine becomes pale and remains thus throughout infancy due to the low specific gravity at this age (1007-1010). It is not uncommon to find sugar in a baby's

urine This does not indicate any failure of carbohydrate metabolism but is due to the fact that babies are apt to have such a high sugar intake that the sugar threshold for the kidney may be passed

**Examination of the Urine** Pyelitis is a very common complaint in babies but the amount of pus which is passed is often very small and the ordinary boiling or nitric acid tests for albumin may fail to give any appreciable cloud of albumin The pus will only be found by microscopic examination of the urine This is a point of real practical importance and no examination of a baby's urine is complete without the microscopical report It is a mistake to centrifuge the urine as an entirely false idea of the number of cells is thus obtained The freshly passed urine should be stirred up merely and a drop put on a slide a coverslip placed over this and the urine examined first under the low power of the microscope and then under the high More than two cells seen regularly per field under the high power will suggest pyelitis Often the field will contain scores of cells and yet the urine only give the faintest cloud on boiling and adding acetic acid In girls it is best to obtain a catheter specimen as a slight vaginal discharge may lead to confusion in the diagnosis

## URINARY ABNORMALITIES OF INFANCY

Transient albuminuria has already been mentioned Its significance is doubtful when unassociated with other findings

Hæmaturia is a common and important urinary condition in the baby During the neo natal period it is most commonly caused by hæmorrhagic disease of the new born (see p 58) rarely by uric acid infarction and rarer still by sepsis

After the fifth month it appears as the first symptom of scurvy It may also be associated with trauma balanitis leukæmia purpura hæmophilia and neoplasm Certain irritant drugs especially hexamine may sometimes cause hæmaturia and the condition should be kept in mind when they are being used

Hæmoglobinuria is sometimes seen associated with acute infection and severe fatal jaundice in the new born

Bile pigments are usually present in all types of neo natal jaundice Other pigments appear occasionally in babies and young children's urine and sometimes cause alarm They are

due usually to the eating of sweets impregnated with certain dyes

**Bladder Control** The age at which bladder control is acquired varies greatly and is often earlier in girls than boys. It depends to a great extent upon the training which the infant receives. A good nurse will sometimes be able to get the baby to control the urine during the day before the end of the first year with others it will be impossible till after the second birthday. If control has not been established both for day and night by the third birthday the condition must be regarded as pathological.

Paralysis of the bladder is not uncommonly associated with spina bifida (both external and occult) and sometimes occurs after surgical removal of the sac. It is also met with as a sequelæ of birth injury in difficult breech cases. The symptoms are usually present at birth. There are two main varieties —

(1) Complete retention and overflow incontinence

(2) Continuous dribbling associated with a paralysed sphincter

The diagnosis is important and often difficult in cases of slight weakness of control associated with spina bifida occulta. In these cases the latter condition should never be presumed to be the cause till therapy has failed. However in the problems of the first year we are only concerned with the severer forms where the diagnosis is usually clear. Treatment of these is very unsatisfactory catheterisation often leading to infection while if nothing is done hydronephrosis tends to follow. In cases of spina bifida occulta surgical treatment (e.g. freeing of the nerves from adhesion or pressure) is sometimes successful.

**Malformations** Malformations of the urinary tract are amongst the commonest congenital deformities.

One or both of the kidneys may be absent or rudimentary. They may be double joined (horse shoe kidney) or have multiple ureters or be movable. Besides these conditions they may show numerous cysts at birth. This latter condition *congenital cystic kidney* is due to dilatation of the tubules. It usually affects both kidneys. the cysts may be small and numerous or few and large. The kidney is usually much enlarged and its function greatly impaired. In severe cases diagnosis may be made during infancy though in the milder cases no trouble will be suspected for some years. then

urinary insufficiency will gradually make itself apparent. Death from uræmia is the usual termination.

The diagnosis may be difficult. A pyelogram may be of great help in these cases though it is difficult to perform on a baby of under one year.

The most important of these congenital abnormalities apart from the severe types which are incompatible with life are those which lead to urinary obstruction and its sequelæ such as hydronephrosis. The latter may be caused by paralysis of the bladder, neoplasm, calculus, malformations of the kidney, ureter and urethra (in boys) as well as stricture.

**Hydronephrosis.** The clinical syndrome here will depend upon the site of the obstruction.

(a) *Infravesical.* This type is seen in male infants and is usually due to a valve flap passing from the verumontanum to the urethral wall. Occasionally complete obliteration of a section of urethra is found. In valvular obstruction the back pressure first affects the bladder which undergoes dilatation and secondary hypertrophy. The condition may thus be compensated for some time but in severe cases this gradually breaks down and the bladder fails to empty completely. Dilatation then takes place followed by incompetence and usually infection. At the same time the back pressure tends to cause dilatation of the ureters and pelves of the kidneys resulting in bilateral hydronephrosis. The clinical symptoms may take some time to manifest themselves or a dribbling incontinence may appear shortly after birth. When the abdomen is palpated a globular swelling will be felt arising from behind the symphysis pubis into the hypogastrium. Sometimes the hydronephrosis can be palpated also. Where trouble is caused by a valve and when the condition is diagnosed early enough it may sometimes be cured by the passage of a catheter though if the valve faces upwards the catheter can be passed into the bladder without permanently relieving the condition. In these cases further surgical measures will be required. The intravesical pressure should be let down slowly as shock associated with anuria may supervene if this is done too quickly. In cases where the obstruction is removed satisfactorily and there has been little or no infection the prognosis is good the bladder and kidneys recovering rapidly.

(b) *Supravesical.* The causes of supravesical obstruction are numerous in infancy, the commonest being due to congenital

malformation of some portion of a ureter. This type of lesion is usually unilateral and results in rapid dilatation of the affected side very commonly associated with infection. The function of the affected kidney may be completely or partially destroyed but few symptoms may appear at first the other kidney hypertrophying and taking on the function of both. Eventually as a rule infection supervenes and the case comes to be diagnosed during the investigation of a chronic pyelitis (see below).

For exact diagnosis special urinary investigations are necessary in these cases. These are naturally difficult to carry out in the baby but with modern technique they are usually possible.

A plain x ray may show the outline of an enlarged kidney. Intravenous pyelography occasionally shows up the condition clearly though under one year this procedure has great technical difficulties. Cystoscopy combined with the use of indigo carmine may be helpful and occasionally proves of real service in the diagnosis.

## THE BLADDER

Malformations of the bladder are not common. The chief types may be summarised as follows —

- (a) *Ectopia Vesicæ* (Exstrophy of Bladder). In this condition there is an absence of the anterior pelvic bones which are replaced by a fibrous band. The bladder itself is represented by a red patch of mucous membrane on the front of the abdomen in the hypogastric region into which the ureters open and from which the urine oozes. The condition is almost solely seen in boys. As a rule the testicles remain in the abdominal cavity the



FIG. 24.—*Ectopia vesicæ*. General view.

ejaculating ducts may be seen in the exposed prostatic urethra and the penis is shortened with a groove along its dorsal surface (see Figs. 24 and 25).

The condition when it occurs in girls is analogous. The vagina is usually absent and the clitoris and labia separated.

Ectopia vesicæ is compatible with life infection of the ureters not readily occurring. Hence every effort should be made to nurture the child until surgical treatment can be attempted. The urine dribbles away continuously, and the child has a permanent ammoniacal smell. The surrounding skin tends to become excoriated unless carefully attended. The exposed mucous membrane may become infected and ulcerated.

Various plastic operations have been elaborated—probably the most useful being that of transplantation by stages of the ureters into the sigmoid colon. There is a certain degree of risk in this procedure as later coliform infection may spread up the ureters from the colon. However this risk is counterbalanced by the advantages of the successful operation for the bowel



FIG. 25.—Ectopia vesicæ. From same case as Fig. 24 showing enfolded mucous membrane with opening of ureters in centre. (Plates by courtesy of Mr. A. B. Clery, Richmond Hospital, Dublin.)

soon becomes accustomed to holding quite large amounts of urine for several hours, thereby allowing the individual to live a more or less normal life. In partial cases reconstruction of the abdominal wall is sometimes attempted.

(6) Patent Urachus. Complete patent urachus with actual discharge of urine from the umbilicus is a very rare condition but minor forms are not so uncommon. The urachus may have a blind external opening at the umbilicus or may form a cul de sac for some distance from the bladder. Sometimes it is patent in the middle and blind at both ends, a cyst forming. Surgical removal of the latter or obliteration of the patent type should be undertaken. Patent Urachus is often associated with obstruction in the urethra.

Infections. These are best divided into those occurring during the neonatal period and those of later infancy and childhood.

## Pyelitis

During the first week of life it is not uncommon to see children with a slight temperature who are passing very little urine. Some of these cases clear up on extra fluid being given and probably belong to the 'dehydration fever' group (see p. 60). If the urine is examined it will be found to contain no pus cells. In another similar group however numerous pus cells, casts and coliform organisms or occasional mixed infections are met with. The common symptoms are reduction of urinary output or anuria, thirst and irritability, vomiting, œdema and sometimes collapse and death. Occasionally generalised convulsions are also associated with the condition.

One of the most interesting points brought out by W. S. Craig in connection with pyelitis at this age is the fact that it occurs more frequently in boys than girls.

The cause of the condition is obscure and more work needs to be done on its pathology. Maternal toxæmia probably plays an important part. Provided the child is not very toxic and does not collapse the prognosis is good though it may be some months before the infection has disappeared completely from the urinary tract. Treatment consists in obtaining free diuresis by means of extra fluids and the administration of sodium citrate gr. x xx per diem. The urine must be made alkaline and if the above dosage of alkali is insufficient it must be increased. In severe cases subcutaneous infusion and the substitution of water and glucose for milk feeds may be necessary for some days.

Pyelitis occurring after the neo natal period has a different ætiology and clinical syndrome. It is one of the commonest pathological states between four months and two years. It is very much more common in girls than boys. Indeed it is rarely seen in the latter unless some congenital urological abnormality is present. The infecting organism is usually the bacillus coli though occasionally other organisms are found. This together with the increased frequency in baby girls suggests that the infection spreads as a rule from a urethral orifice which has become infected from the feces. It is probable however that infection occurs by way of the blood stream in certain cases particularly when the organism is other than the bacillus coli.

Pyelitis in infancy is very often associated with gastroenteritis and sometimes with respiratory infection.

**Pathology** It is difficult to obtain exact information in acute early pyelitis as cases of the disease that come to autopsy are usually of old standing. It appears however that the pathological condition is chiefly one of catarrhal inflammation of the pelves of the kidneys together with some degree of inflammation of the calyces and some associated cystitis. The inflammation of the bladder seldom causes symptoms in babies. Pyelonephritis may be slight or severe. small abscesses may be found in the kidneys. In long standing cases of pyelitis the mucous membrane becomes almost entirely destroyed. In cases diagnosed early and treated successfully the condition clears up completely and leaves no sequelæ. In severe cases associated with some degree of pyelonephritis however the kidney substance may be severely damaged and lead to renal insufficiency in later life.

**Symptoms** These are very variable. sometimes there is nothing but a slight elevation of temperature for a few days, some loss of weight and anorexia and then recovery. In others there may be a high swinging temperature, pallor, rapid loss of weight or collapse. If undiagnosed the case may become chronic and the baby be brought to the doctor for failure to gain weight or marasmus. The urine is usually highly acid and often concentrated. it may contain large numbers of single pus cells or these may be in clumps. often epithelium cells and casts are present as well and sometimes red blood corpuscles. Often there is no correlation between the amount of pus and the severity of the general symptoms. The temperature may be high and the baby very ill for some days and yet the urine contain few if any pus cells. then suddenly it becomes crowded with cells while the temperature falls to normal. As a rule coliform bacilli can be seen in the urine and grown in pure culture from a catheter specimen.

In babies bladder symptoms are rare though crying and irritability often suggest pain. The onset is often associated with vomiting sometimes with convulsions but seldom with rigor as in adults. In most cases there is a leucocytosis of 15 000-30 000.

The course of the disease is very variable—from one week to many months. If treatment is efficiently given and the condition does not clear up some congenital abnormality should be suspected.

**Diagnosis** Pyelitis is not a difficult disease to diagnose, yet it must be one of the most frequently missed. Only in those cases in which the pyuria is intermittent is there any excuse for this however. In every case of illness in a baby the urine should be obtained and examined. As we have already pointed out the ordinary chemical tests are not sufficient, and the urine must be microscopically examined as well. Every pædiatrician has had the experience of having been called in consultation on a case of obscure fever in which there were few if any signs, but the child was running a high temperature and appeared ill. The doctor said the urine contained nothing but when questioned admitted that he had not examined it microscopically. A fresh specimen was then obtained and a drop placed under the microscope and found to contain pus. One word of warning is necessary: other sources of pus must be ruled out. In male children pus cells may be discharged into the urine from an infected prepuce; in females from a vaginal discharge.

In chronic cases which fail to clear up upon treatment, malformations must be looked for and pyelography or cystoscopic examinations may be necessary.

**Treatment** In all cases of acute pyelitis *alkali treatment* should be given a fair trial and other measures only resorted to when it fails. This treatment will be successful in the vast majority of cases if administered correctly and has the advantage that it can be continued over a considerable period of time and is easy to give. It is of course useless in cases of obstruction. The essential features of the treatment are (1) to obtain diuresis, (2) to make the urine alkaline. The best method is to increase the dosage of alkali rapidly till the first morning specimen gives a pH 7.6. The actual amount of alkali necessary will depend upon the age and size of the baby. Every baby is different in this respect. Some babies of six months will require sodium citrate gr. x four hourly, others gr. xx others still more.

If the alkali treatment fails acid therapy should be given. There are a number of different methods of doing this. The oldest consists of combining hexamine with acid sodium phosphate. More recently the *ketogenic diet* has been introduced. This consists essentially in reducing the carbohydrate intake and increasing the fat till a marked ketosis is produced. More recent still, as a substitute for *l*-oxybutyric acid, the supposed active principle in the ketogenic diet, *mandelic acid*

has been introduced. The latter is combined with ammonium chloride to make the urine acid.

In babies the ketogenic diet is impracticable for before enough ketosis has been produced the child will be suffering from a fat dyspepsia. In children over two years the present author has used it with success in some half-dozen cases. The essential feature of all these treatments appears to be to produce a sufficiently low pH while supplying some urinary disinfectant at the same time. We have found that it is usually necessary to produce a pH 5.2 before beneficial effects occur. Then sometimes the treatment acts like a charm the case clearing up in a few days. Acid sodium phosphate except in infancy is not sufficiently powerful an acid and ammonium chloride or other powerful acid salt should be given whether hexamine (urotropine) or mandelic acid are to be used as the germicide. Here again the dosage is variable. Hexamine gr 4-11 five times a day may be given to a child of six months or mandelic acid gr 1x per diem. Both being increased as the child grows older. The dosage of ammonium chloride is more variable. The object is to obtain a constant urinary pH 5.2 or less. Ammonium chloride gr xl per diem should be sufficient for a baby of six months given in five doses during the twenty four hours. Some cases will require more and often the child will be inclined to vomit before a sufficiently large dose is reached.

Recently the introduction of ammonium mandelate has greatly simplified treatment more particularly during infancy and childhood. Payne recommends the following dosage —

Ammonium mandelate under six months	gr 30
two years	gr 30-60
five years	gr 60-90

The dosage will vary in every case and can only be decided upon when the urinary pH has been estimated after the child has been on treatment for some days. In certain cases additional acid salt will have to be added to reduce the pH sufficiently. Payne recommends the following mixtures for children —

*Mistura Ammonii Mandel*

Ammonium mandelate	gr 20
Liquid extract of liquorice	℥ 5
Elixir of gluside	℥ 1
Water	ad 5 1

*Mistura Ammonii Phosphas*

Ammonium phosphate	gr 7½
Liquid extract of liquorice	℥ 5
Acid syrup	℥ 15
Water	ad 5 1

These substances may act as irritants to the kidney and may cause hæmaturia and casts may be found in the urine. They should not be given for longer than weekly or ten daily periods at a time when they should be stopped and alkali treatment instituted again. Sometimes if the changes are rung in this way the condition will clear up when the diuresis sets in with the renewal of the alkali treatment.

In all the acid treatments it is wise to limit the consumption of fluid so as to increase the concentration of the urine.

Certain American writers claim excellent results from intravenous injections of mercurochrome and arsphenamine but we have had no experience of therapy with these substances. In a number of persistent cases however we have felt that great benefit has resulted from the use of autogenous vaccines.

Recently we have used prontosil with success in a number of resistant cases.

**Perinephric Abscess**

This condition results from cellular inflammation of the tissues surrounding the kidney and may be found at any age though it is exceedingly rare in babies. The earliest recorded case is in a baby one week old. The symptoms are essentially the same at this age as in later life but may be difficult to elicit. The leg on the affected side is usually held flexed extension causing pain. There will be elevation of temperature pain associated with crying loss of weight anorexia and general malaise. There may be local tenderness over the kidney area from behind but this is difficult to elicit in babies. A scoliosis towards the affected side will be found pain resulting if an attempt is made at correction. A leucocytosis is present in all cases.

The inflammation may subside without abscess formation or an abscess may form and point in the ilio costal region or in the iliac fossa.

Diagnosis in infancy may be very difficult unless local swelling develops in the vicinity of the affected kidney.

Treatment consists in poulticing the affected area till

diagnosis is reasonably certain when surgical drainage should be carried out

### Urinary Calculi

These occur fairly frequently in infancy especially in certain districts. The commonest consist of uric acid and are seldom larger than a pea. Frequently they are multiple and small often being passed per urethra. They are due to the high uric acid concentration in the urine in early life and tend to disappear in later childhood. Large calcium phosphate stones are occasionally met with but are very rarely seen under one year. The small uric acid stones of infancy do not appear to damage the kidney nor do they cause any symptoms as a rule. The passing of a stone or a number of small stones (urinary gravel) however may cause hæmaturia and agonising pain will be complained of in children old enough to speak. Small infants will sometimes scream uncontrollably for some minutes before passing sand or a small stone.

Large stones lodging in the bladder are said to be associated with a *B. proteus* infection and an alkaline urine though we have never seen such a case in an infant. Very rarely calculi cause urinary obstruction (see *Hydronephrosis* above).

Stones seldom form in the bladder unless there is some degree of obstruction to its outlet. In the absence of such obstruction the majority of stones found in the bladder have been previously formed in the kidney and passed down the ureter into the bladder subsequently. This must be remembered when dealing with the condition of vesical stone *e.g.* it is useless merely to remove a stone from the bladder when the real trouble is in the kidney.

### Acute Nephritis

Nephritis is very rarely seen under one year of age. Its ætiology and pathology do not differ at this age from those seen in later life. The subject cannot be dealt with briefly. Therefore we have preferred to omit it from this work and refer our readers to the general text books of medicine for descriptions of the condition.

### Genital Organs

Malformations of the male genital organs such as phimosis, etc., are dealt with on p. 357.

The only malformations in the female genitals of importance in this work are atresia of the vulva and imperforate hymen. The former is caused by adhesions which are seldom dense, and can usually be broken down at birth without an anæsthetic. The latter requires opening by simple incision, so as to prevent the accumulation of mucoid secretion and menses in later life.

## CHAPTER XVIII

W R F COLLIS

### DISEASES OF THE DUCTLESS GLANDS

(The Thyroid Gland—Cretinism Symptoms Diagnosis Treatment—Hypothyroidism—Hyperthyroidism Symptoms Treatment—The Pancreas—The Parathyroid Glands—Hyperparathyroidism—Suprarenal Glands—Hypernephromata—Congenital Neuroblastoma—Addison's Disease—The Pituitary Gland)

ENDOCRINOLOGY is in the forefront of investigative medicine at present. Much new light has been thrown recently on the function of the pituitary gland whose internal secretions are now regarded by some as the time keepers for all the other ductless glands. There is no doubt that the internal secretions have a profound effect upon growth and development during infancy and it is probable that as we come to know more of their mode of action we will be able to diagnose and treat certain cases which now are designated 'marasmus' for lack of a more definite term.

#### The Thyroid Gland

We have already dealt with the part played by the thyroid gland in prematurity (see p 51) and therefore will confine ourselves here to its function after the neo natal period.

#### Cretinism

This condition depends upon absence or insufficiency of the internal secretion of the thyroid gland. It is regarded as a congenital condition and due to failure of the gland to develop. Post mortem examinations on babies dying with the condition have shown usually absence of the thyroid gland.

The condition tends to occur sporadically in single members of different families and does not appear to be hereditary or apt to occur in more than one child of any family.

**Symptoms** The symptoms depend upon the degree of

the condition in any case they do not appear as a rule till the second half of the first year, mild cases often pass undiagnosed till the third or fourth year. What causes this delay in the first appearance of symptoms is not clear. The suggestion that the mother supplies the child *in utero* with sufficient thyroxin to carry it on for the first six months is hardly borne out by the fact that the normal baby's thyroid gland commences to function shortly after birth.

The most characteristic feature of the condition is failure of growth and development. The child becomes thickset and podgy. The limbs are short compared with the trunk, the hair is scanty, the fontanelles remain open, the palpebral fissures are slit like, the nose is flat, the tongue is broad and flat and appears too big for the mouth, the subcutaneous tissues are thickened, the abdomen is pendulous. The temperature is subnormal and the basal metabolism low, hence cretins often suffer from cold. As a rule they are good natured and little trouble. Persistent constipation is sometimes a marked feature. Their general appearance is very characteristic the baby appearing dull backward and almost mentally deficient and having a coarse dry skin.

**Diagnosis.** In a pronounced case the diagnosis is obvious. Confusion however, not uncommonly occurs between cretinism and mongolism. Below is given a table of differential diagnosis between the two conditions —

	Cretinism	Mongolism
<i>Commencement</i>	Second half of first year	Birth
<i>Mental state</i>	Lethargic	Backward
<i>Temperature</i>	Subnormal	Normal
<i>Congenital at normal times often present</i>	Unilateral hernia	Congenital heart disease
<i>Skin</i>	Dry	Not characteristic
<i>Subcutaneous tissue</i>	Thickened abdomen pendulous	Not abnormal
<i>Eyes</i>	Wide apart and palpebral fissures slit like	Epicanthic fold specially developed, Mongoloid appearance
<i>Joints</i>	Normal	Flaccid
<i>Fingers</i>	Stubby and short	Little finger characteristically half size of ring finger
<i>Onset of reaction to treatment with Thyroid</i>	Retarded Marked	Not changed None

**Treatment** If diagnosis is made early and treatment commenced forthwith, the baby usually becomes normal though treatment must be continued for life and the person may always be stupid and slow. The reaction to treatment is rapid and marked. The constipation clears up, the temperature becomes normal the skin loses its coarseness and the general torpid condition gives way to the healthy state of the normal child.

It is always well to commence with a small dose such as thyroid extract gr  $\frac{1}{2}$  (B.P.) twice a day and then increase till symptoms of irritability and looseness of the stools begin to appear. The dose is then reduced just sufficiently to allow these toxic manifestations to disappear and then maintained at that level.

### Hypothyroidism

Distinct from sporadic cretinism where there is congenital lack of secretion of the thyroid gland a group of cases with symptoms of mild hypothyroidism are met with occasionally. These cases are seldom seen before the second or third year, though it is probable that some commence before that date. Their symptoms are delayed ossification thick lips enlarged tongue, slow mental development—in fact general slight retardation. The diagnosis rests to some degree upon the reaction to treatment with thyroid the genuine cases of hypothyroidism improving while the others fail to respond to therapy.

### Hyperthyroidism

A few cases of hyperthyroidism occurring during infancy, including one congenital case are recorded in the literature but the condition is so rare at this age as to be of academic interest only.

*Goitre* or hyperplasia of the thyroid gland is not uncommonly found in babies during the first year of life in districts where the disease is endemic. We have seen during the past year, two cases of congenital colloid goitre in babies born of mothers suffering from the same condition.

The disease is thought to be due to lack of iodine in the soil and hence also in the water supply of certain districts. An infection is also said to play a part in the production of the disease. Infants suffering from goitre are usually the offspring of goitrous parents on one or both sides of the family.

**Symptoms** In congenital cases the thyroid gland is usually involved in its entirety showing general hyperplasia without cyst formation. Sometimes the tumour is so large as to obstruct the birth. Sometimes it obstructs respiration and leads to asphyxia following attacks of dyspnoea. Occasionally the condition is associated with decreased function of the gland and cretinism occurs.

**Treatment** The preventive treatment of goitre is to ensure a satisfactory and pure water supply. This can be done by adding iodine in small quantities to a central filtered water supply or by boiling the well water of goitrous districts and supplying the inhabitants with iodine from time to time.

In the early stages of the disease iodine will sometimes cure the condition and should be given to all cases diagnosed during infancy. If symptoms of hypothyroidism appear thyroid extract must be given without delay.

### THE PANCREAS

Certain specialised cells (the Islands of Langerhans) in the pancreas produce an internal secretion insulin which largely controls carbohydrate metabolism in the body. Loss of function of the islands leads to insufficiency of insulin secretion and the clinical condition of *diabetes mellitus*. Though extremely rare under six years of age diabetes has been described in babies only a few months old.

The aetiology of the disease is obscure. In about one third of the cases there is a family history of the condition on one or the other side of the family. Cases of glycosuria at birth have been reported but we have never found any symptoms of the disease in babies of diabetic mothers during the neo natal period. Sometimes the onset follows an acute infection such as gastro-enteritis more often when diagnosed no predisposing cause can be found.

**Prognosis** Before the introduction of insulin therapy all babies and indeed all young children contracting the disease died in a short time. Now with proper care even in those cases occurring during infancy the prognosis is not hopeless though the younger the child the more difficult the management of the case.

**Symptoms** The onset is usually acute in the baby and is associated with loss of weight failure to thrive crying due to

thirst, polyuria (causing scalding of the buttocks and thighs), pruritis and possibly secondary infection, *e g*, furunculosis

The diagnosis of diabetes in the infant is difficult and must not be made solely on the evidence of sugar in the urine. Babies fed on a high sugar formula not uncommonly pass sugar in their urine. Ketonuria is also common at this age. Hence it cannot be too firmly impressed upon doctors that no baby should be diagnosed as diabetic and given insulin before a blood sugar estimation has been made. Several excellent micro methods have now been worked out which allow the estimation to be done on less than 0.5 c.c. of blood.

**Treatment** The treatment of diabetes during infancy will need the constant co-operation of the physician and biochemist if the correct dosage of insulin is to be maintained. The baby during the first year is perforce on an almost exclusively milk diet, though during the second six months other constituents are being added gradually. If the child is being fed correctly at this time *e g*, is receiving 45-50 calories per pound of body weight per day (see Infant Feeding p. 91) it will not be possible to reduce the feeding formula if the child is to thrive, nor is it wise to reduce relatively the amount of carbohydrate. Infants usually tolerate fat badly and any increase is apt to cause fat indigestion and ketosis. Therefore as soon as the condition is diagnosed and the blood sugar worked out insulin therapy should be begun without delay. Babies require relatively larger doses of insulin than adults, due to the fact that they are receiving relatively much larger amounts of sugar. In the baby insulin reduces the blood sugar with great rapidity, but it tends to rise equally quickly again. Hypoglycæmia is always a danger in the baby or child. Constant urinary examinations and blood sugar estimations are necessary during the initial stages of the treatment, and it is important to have the child under careful observation, and if possible in hospital, during this period. The dose of insulin depends upon the case, each being different. It is always well to start with a very small dose such as 1 unit, and estimate its effect before increasing. In babies three or more doses of insulin should be given per twenty four hours. The objection that older children and adults have too numerous hypodermic injections does not hold at this age and it is easier to control the blood sugar if the number of injections is increased and the amount of insulin per dose decreased.

Infections lead to temporary exacerbations of the condition and are always dangerous in diabetic infants. Constant urinary examinations and blood sugar estimations will be necessary if the baby is to be steered through these dangerous periods. Every care must be taken to isolate diabetic babies from intercurrent infection of any kind. If they are in hospital they should be kept in a single ward and if at home they must be isolated as far as possible.

Hypoglycæmia associated with convulsions and unconsciousness occurs sometimes without warning and may be confused with coma. The urine should be obtained and tested at once. In diabetic coma the urine will contain ketone bodies and sugar in hypoglycæmia neither. A blood sugar estimation should be made as quickly as possible and the child given glucose (5 per cent.) intravenously at once.

### PARATHYROID GLANDS

The Parathyroids are minute endocrine glands situated either in or close to the thyroid gland and are necessary to life. Their internal secretion parathormone regulates the amount of calcium in the blood serum by controlling its liberation from or lying down in the bones. It does not control absorption or excretion of calcium these functions being regulated by vitamin D. Removal of the parathyroid glands gives rise to the clinical syndrome of tetany. The common form of tetany found during infancy and childhood is now thought to be due to a rachitic condition (caused by a vitamin D deficiency) occurring in association with a hypoparathyroidism. The matter is more fully discussed elsewhere (see p. 125).

The many problems of calcium metabolism in connection with pregnancy, nervous conditions (e.g. chorea), eye conditions (e.g. lamellar cataract) etc. are receiving much attention at present though as yet the exact positions that parathormone and vitamin D play in relation to the different syndromes has not been fully worked out. Occasionally tetany occurs in association with cretinism the parathyroid glands being deficient as well as the thyroid gland. In these cases the tetany can be treated by the administration of parathormone.

Hyperparathyroidism produces the clinical syndrome known as *generalised osteitis fibrosa*. There is absorption of calcium from the bones together with the formation of multiple foci

cortex on the other hand produces an internal secretion which plays an important part in sexual differentiation and development. Study of cases of hyperplasia and tumour of the cortex have established the fact that hypersecretion produces sexual precocity in the male and virilism in the female and occasionally curious cases of apparent hermaphroditism.

**Addison's disease** hypofunction of the suprarenal glands. The disease rarely occurs in children and still more rarely during infancy and hence readers are referred to the text books of general medicine for descriptions of the clinical syndrome of this condition. Recently interest has been aroused by claims of beneficial effects by injection of suprarenal cortical extract in certain cases of marasmus.

**The Pituitary Gland** We have already alluded to the fact that the pituitary is now regarded as the time keeper for the other ductless glands producing a large number of internal secretions. As this work is only in its infancy and as the present known pathological states which are associated with hypo and hypersecretion of the different parts of the gland are not described as occurring in infancy no description is necessary here.

**Disease of the Thymus Gland** (see p. 425)

## CHAPTER XXIV

W R I COLLIS

### INTESTINAL WORMS

IN Ireland there are three common types of worm found in children thread worms round worms and tape worms Round worms and tape worms are rarely if ever met with during infancy hence only the thread worm need be considered here

#### Thread Worms (*Oxyuris Vermicularis*)

This is the commonest type of intestinal parasite found during infancy and childhood The worm varies in length from  $\frac{1}{2}$  to  $\frac{3}{4}$  of an inch is wider at one end and resembles a piece of grey or white thread It inhabits the large intestine and is often found in the appendix where some authorities believe it breeds others hold that the ovum cannot develop into the mature worm without leaving the large intestine At night the worms not uncommonly wriggle out of the anus and set up irritation in the surrounding parts The ova when passed often stick to hairs or to the skin of the buttocks and if the child scratches himself and then sucks his fingers he is apt to re infect himself The ova may be conveyed from one child to another in water milk or fruit flies acting as carriers

Symptoms associated with thread worm infection are very indefinite In a healthy child the infection is often transitory and almost symptomless—perhaps there is some mucus in the stools or some slight local irritation but nothing more It is probable therefore that such symptoms as tiredness lack of vigour, debility constipation colitis nervous irritability etc which are often ascribed to thread worm infection are rather the cause than the effect More likely the child is in a debilitated anæmic and constipated state and hence the parasites have found a suitable host in which to thrive The principal symptom is local irritation around the anus and genitalia which may be very severe and lead to inflammation of the surrounding parts This may cause frequency of micturitis in the male and vaginitis in the female

worms are present in large numbers there may be pain on defecation from time to time associated with the passing of a stool containing much mucus and numbers of parasites

Treatment must first take into account the child's general state of health. If he is debilitated and constipated his general health must be attended to all foci of infection such as bad teeth and septic tonsils being eliminated the diet corrected and the constipation treated before local measures to eradicate the parasite are likely to prove altogether successful.

The worms may be found in any part of the large intestine and are often met with in considerable numbers in the cæcum and appendix. For this reason it is well to combine treatment by mouth with enemata. The most useful drug is santonin (also used for round worms) and the following mixture as recommended by the Pharmacopœia of the Hospital for Sick Children Great Ormond Street is valuable (the dosage being varied to suit the age of the child) —

Santonin	gr 1½
Compound powder of scammony	gr 2
Calomel	gr 1

Enemata may be of simple saline or infusion of quassia. Bichloride of mercury 10<sup>-1</sup> to 10<sup>-2</sup> is probably the most efficacious injection and may be repeated every other night till the parasites have disappeared. The fluid should be injected as slowly as possible and retained as long as possible—in babies the buttocks will have to be held together.

The child must be prevented from re-infecting himself scrupulous cleanliness being observed. A mercurial ointment should be smeared round the anus to prevent the parasites from coming out at night. Babies should be placed in restrainers to prevent them scratching.

In mild cases these measures will be rapidly successful but in severe cases where the worms are present in great numbers and are high up in the cæcum and where the child's general health is poor it may be a long time before the cure is complete.

## SECTION V

### CHAPTER XXV

H L PARKER

#### DISEASES OF THE NERVOUS SYSTEM

(Methods of Examination of the Nervous System in Infancy—Developmental Disorders—Congenital Hydrocephalus—Anencephalus—Microcephalus—Acrocephalus—Mongolism—Tuberous Sclerosis Epilola—Klippel Feil Deformity—Mental Deficiency in Infancy—Cerebral Palsies of Childhood Cerebral Diplegia *Infantile Hemiplegia*—*Encephalitis of Infancy*—Diseases Affecting the Cerebro-spinal Vascular System—Amaurotic Family Idiocy—Intoxications and Deficiency Disorders—Pink Disease )

#### Methods of Examination of the Nervous System in Infancy

THE main difference between an infant and an adult in terms of examination is that the former is incapable of the same degree of co-operation as the latter. Nevertheless just as accurate information may be obtained if the examination is performed in the proper way.

(a) The child should be uncovered completely in a warm room and time allowed for him to recover from the excitement of being undressed.

(b) The child should not be touched until he has been observed carrying on the normal activities of an infant at rest. The size and shape of the head can be noted the movement of the eyes facial expression and response to movement in the immediate vicinity. Muscular activity under normal conditions is constantly in play and these movements should be watched closely for evidences of abnormality. Probably the most important thing to watch for is the attempt on the part of the child to enlarge his sphere of co-ordinate activity. Grasping at objects, playing with his toes, attempting to hold his head up and stuffing objects into his mouth are all signs of normal development. It is of no use to try to watch for these signs in a crying fretful baby and time and patience are required until peace is restored.

(c) When the child has become accustomed to the examiner's

presence gentle and slow movements may be made to determine more closely his physical condition. *The skull and fontanelles* should be palpated and *rigidity of the neck* excluded. With patience and care the *optic disc* can be seen with an ophthalmoscope and the pupil reflexes investigated. If the infant is old enough *ocular movements* may be determined by passing a brightly coloured object or a light in front of his face. *Weakness of one or other side of the face* is easy to recognise, and some response can be obtained by placing a watch to the child's ear as a test of *hearing*. *The bulbar mechanism* comes into play during feeding and abnormalities can then be noted. *Tendon reflexes* in a small infant are hard to obtain but by using a small rubber covered percussion hammer and waiting for the most favourable moment of relaxation the examiner will find little difficulty in obtaining the biceps patellar and achilles reflexes. The response to stimulation of the sole of the foot of an infant during the first two years of life is usually extensor in character as far as the great toe is concerned and accordingly an extensor response must not be regarded as abnormal. More important however is the determination of the presence or absence of the normal withdrawal reflex as when the child rapidly pulls the foot and leg away from the stimulus. *The tone* of the muscles in the extremities can be palpated while quietly handling the baby and any undue rigidity or flaccidity may be demonstrated. On turning the child over on his face the spine can be inspected and palpated so that spina bifida occulta or cystica will not be missed. The condition of the *anal sphincter* can be seen and in case of doubt its tone can be tested with the point of the little finger. Presence or absence of an anal reflex can be determined on stroking the anal region with a wooden applicator and in the same way the *abdominal reflexes* may be brought out and the *cremaster* in the case of male infants. These superficial reflexes are usually very brisk active and easily obtained in small infants. With the child on his face observations may be made of his efforts to raise the head draw up the legs crawl or turn over on his back. *The testing of sensations* is not an insuperable task. A normal infant will make some response to light touch or tickling showing that he feels the stimulus. Testing for the presence or absence of *pain sensibility* especially in a condition such as spina bifida with paralysis requires special technique. It should

be the last thing done to the baby during an examination for it commonly breaks all friendly relationship with the examiner. For the test a pin with a large glass head can be used. The child should first be prodded several times with the head of the pin, then a slight prick with the sharp point can be slipped in between the innocuous stimuli, whereupon a change in the child's attitude will immediately take place. It is needless to add that once pain has been induced, crying follows and the examination to all intents and purposes is at an end.

### Developmental Disorders

Considering the complicated processes involved in the development of the nervous system, it is little wonder that frequently errors of development occur. How many of these defects are due to hereditary characteristics transmitted in the genes and how many are due to trauma, disease of the mother or foetus or mere accidental failure of development, is always a matter of speculation.

**Congenital Hydrocephalus** In infancy the bones of the skull are soft, ununited at the sutures, and, consequently, in contrast to that of the adult, the head has the capacity for considerable enlargement. An increased volume of cerebrospinal fluid with increased pressure in the ventricles may lead to this enlargement. This in turn may be due to a disturbance of the formation, circulation, or absorption of the fluid. The cerebrospinal fluid is formed as a filtrate by the choroid plexuses of the cerebral ventricles, passes through the ventricular system and reaches the subarachnoid space by the foramina of Magendie and Luschka. From there it passes to the surface of the brain and spinal cord where it is absorbed into the blood stream by the arachnoid villi of the intracranial venous sinuses and possibly also by the capillaries of the nervous system. Hydrocephalus may therefore be caused by increase in the production of cerebrospinal fluid, a block somewhere along its paths of circulation, or deficiency in its absorption. An increased filtering activity of the choroid plexuses may be due to kinking of the vein of Galen or any other factor that causes a rise of pressure in the vessels of the choroid plexuses. Inflammatory processes or tumours may obstruct the free exit of fluid through the foramina of Magendie and Luschka, or at any part of the ventricular pathway anterior to these openings. This has been called obstructive hydrocephalus. Communicating hydro

cephalus is the condition in which free communication between the ventricles and the subarachnoid space exists and the hydrocephalus is due either to disturbance in the formation and absorption of the cerebrospinal fluid or to an obstruction in the subarachnoid space itself

The division of hydrocephalus into *congenital and acquired types* is too arbitrary for the causes of many cases appearing after birth actually have their inception during intra uterine life. Intra uterine meningitis, ependymitis and developmental defects of the iter of Sylvius are frequent factors in infants born with evidence of hydrocephalus. In rare cases tumours are found present that must have existed before birth. *Common post natal causes* are *meningitis*, *syphilis* and *meningeal hæmorrhage due to birth trauma*. Tumours appear more frequently in older children. In congenital hydrocephalus enlargement of the head may cause difficulty during parturition. On the other hand the child may be born normally with a slightly enlarged head which then progressively increases in size.

**Symptoms.** As mentioned before there may be an associated spina bifida cystica. The head may reach a very large size measuring as much as 30 inches in circumference. The cranial sutures are widely separated the anterior fontanelle is much enlarged and there is marked congestion of the veins of the scalp. The enlargement of the head occurs in all diameters and the orbits are depressed downwards. In some cases only the upper rim of the iris is visible above the lower eyelids like a setting sun and this with an overhanging forehead is quite characteristic. While the condition is increasing vomiting, screaming and convulsions may occur. Optic atrophy and blindness are usual due to pressure upon the optic nerves. Other cranial nerves may become paralysed and squint is not uncommon. Aystagmus and a marked spasticity of the lower extremities have been observed. In severe cases the weight of the enlarged and water filled skull is sufficient to interfere with the child's movements. The mental condition of these children varies considerably and is not altogether in proportion to the degree of hydrocephalus.

The prognosis in this condition is extremely variable and depends largely on the degree of imbalance of the circulation of cerebrospinal fluid. Many cases are quite mild and a balance may be re-established leading to the survival of a child who is left with a large head as the only relic of the condition.

in after life In the majority of cases of hydrocephalus however, the enlargement of the head proceeds intermittently to a fatal termination some weeks or months after birth

**Treatment** In recent years attempts have been made to cure the condition by surgical intervention Procedures such as removal of the floor of the third ventricle extirpation of the choroid plexuses perforation of a blocked Sylvian aqueduct and opening up the roof of the fourth ventricle have been attempted Generally speaking the results have been unsatisfactory, and as yet in the moderate and severe cases of chronic progressive hydrocephalus of infants a cure is lacking In mild cases ventricular or lumbar puncture may carry the patient through to a re establishment of cerebrospinal circulation

**Anencephalus** This is a condition mainly of pathologic interest At birth the calvarium is absent and there are no cerebral hemispheres The nervous tissue at the base of the skull is covered over only by fibrous connective tissue The essential nature of the condition is that for some reason or other during intra uterine life the cerebral hemispheres failed to develop Infants so affected frequently die *in utero* or shortly after birth At the most they may live only a few days

**Microcephalus** During foetal life certain factors may prevent a full development of the brain This may be due to an inherited stigma or to some unknown process interfering during foetal life with the brain's normal development For this reason throughout life the head is unusually small and the child is mentally defective Such cases vary between complete idiocy and feeble mindedness At birth there may be noticed no marked disproportion between the size of the child's head and its body later the disproportion becomes quite conspicuous By the sixth month when normally the head should have grown from about 13 inches to 16 inches the microcephalic head is still only 13 or 14 inches in circumference At twelve months it may be only about 14 inches instead of 18, at two years 15 to 16 inches instead of about 19 The early closing of the fontanelle is an important point in the diagnosis There are often associated convulsions and while there are no real paralyses the limbs of the infant are somewhat more spastic than normal By the second year the so called 'true' microcephalic can be recognised not only by the size of the head but also by its characteristic shape There is a narrow forehead receding frontal parietal bones a pointed vertex and

flattening of the occiput. The chin recedes, the ears stick out and later the nose becomes more prominent. As mentioned before the fontanelle becomes closed either before birth or shortly afterwards. The recognition of the concomitant feeble mindedness and the estimation of its degree will be dealt with in the discussion of amentia due to other causes.

The pathology is characterised by a general diminution of the volume of the brain and this is so marked in the frontal and occipital regions that the cerebellum remains uncovered by the cerebrum. The size of the skull is dependent on this hypoplasia of the brain. The cerebral surface shows a simpler arrangement of the convolutions than usual but the sulci are well marked.

**Acrocephalus.** This condition should come rather under the heading of congenital deformities of the skull. Central nervous system changes are secondary. There is premature closure of the cranial sutures before birth interfering with the normal development of the brain which is often inhibited. The head is abnormally tall, broad and short from before backwards—the *dome shaped skull* (tower skull). There is exophthalmos, the palpebral fissure is oblique so that the external canthus is at a lower level than the internal. Other abnormalities such as arched palate and fusion of the fingers are usually present. Accessory nasal sinuses are rudimentary or absent and there is underdevelopment of the upper and lower jaws. *Clinically the lofty skull and the exophthalmos are the characteristic features.* A small skull may result in increased intracranial pressure so that impairment of hearing, smell and sight with headaches generally occur. Because of the marked protrusion of the eyes there is a stretching of the optic nerves and a resultant optic atrophy.

Skiaographs show prominent digital markings of the inner table of the skull and definite abnormalities of the orbital floors and sphenoidal ridges. The mentality of the child is seldom affected.

**Treatment.** With a view to giving more room for the brain to expand bitemporal decompression has been done and enlargement of the optic foramina has sometimes been suggested but at best such measures are of doubtful value.

**Mongolism.** This curious elemental defect has attracted considerable attention but up to date the ætiology is not completely known. A number of cases of identical twins being

born with this condition have helped to establish the hypothesis that the *defect is in the germ plasm* rather than due to extraneous factors during foetal life. The old hypothesis that the number of children born before the appearance of a Mongolian child had something to do with its causation and that the disease was in the nature of an *exhaustion product* has not stood the test of rigid statistical study. There is however a direct *correlation between the mother's age and the frequency of the affliction*. Children born of women already over forty years of age have a greater tendency to Mongolism though the disease may appear also in the children of younger women.

**Signs and Symptoms** *The head is small rounded and flattened at the back. The eyes have a Mongolian slant such that the outer canthus has a higher level than the inner—hence the fancied resemblance to the Mongolian race. The epicanthic fold of*



FIG. 26. Mongolian Illness. Faces and short incurved fifth finger are visible.

skin at the inner canthus is strongly developed and the nose is broad and flat. *The palate is usually high and narrow and the tongue is prominent. About the sixth or ninth month of life the papillae of the tongue become enlarged and the surface of the tongue has a raw granular appearance. After the first few years of life deep transverse fissures appear on the dorsum of the tongue apparently the result of continuous tongue sucking. The fingers are usually thick for the size of the hand, the thumb is short and the little finger characteristic in that it is dwarfed and curved towards the ring finger. There*

is a complex series of *lines* on the palms of the hands like those on a piece of paper which has been crumpled. The general musculature is hypotonic due to the laxity of the ligaments the joints can be easily hyperextended. *Associated features* are frequently blepharitis coryza and bronchitis and many congenital abnormalities may be found the commonest being congenital heart disease.

There is usually a *retardation of voluntary muscular movements and of mental development*. The infants are not able to support the head until the sixth or ninth month and are seldom able to sit alone before the end of the first year or later. On the average they do not learn to walk before the third year and the muscular co-ordination of both arms and legs is clumsy and feeble. There is delay in acquiring speech which may remain indistinct and limited in degree. The mental and physical development of Mongolism differs little from the less severe types of amentia. The difference is that as a rule Mongols are *lively happy imitative and affectionate*. For this reason the mother usually claims that the child is the best child I have ever had. The mental age however seldom reaches above five years. The ultimate prognosis in these children is not good. Two thirds of them die during the first year of life from pneumonia following the diseases of infancy. Tuberculosis accounts for most of the remainder and very rarely do these children reach puberty or adolescence. The few who live beyond forty become prematurely aged.

The diagnosis can be established at birth but some caution must be observed when informing the parents until signs of mental deficiency are obvious. Mongoloid facies are seen in some individuals with normal intelligence so that it is possible that abortive forms exist. Treatment with thyroid extract has been attempted but nothing improves the condition. (For Diagnosis from Cretinism see p. 258.)

**Tuberous Sclerosis Epiloia.** This is a rare condition and of more interest from the pathological than from the clinical standpoint. The three cardinal symptoms are —

- (1) *Mental deficiency*
- (2) *Convulsive seizures*
- (3) *Sebaceous adenomas of the face*

These appear in the first few years of life and before the age of ten

At birth and during infancy it is impossible to make the diagnosis for the mental deficiency and fits found in the condition differ in no way from those associated with other conditions. There is a strong heredo familial tendency in that among the relatives there may be cases of adenoma sebaceum alone or combined with epilepsy with or without associated mental deficiency. Associated with the condition there is occasionally tumour formation in other organs. Congenital tumours may be found in the heart kidneys lungs spleen and retina. At the age of three or four the combination of epilepsy and idiocy dating from birth and the sebaceous tumours spread in a butterfly fashion over the face is characteristic of the disease. The majority of these children however die in early childhood.

*The pathology of the disease is characterised macroscopically by firm pearly white nodules on the external surface of the brain which project into the third and lateral ventricles. The nodules are more numerous in the cortex than in the white matter. The gyri are large and firm when examined. Microscopically the characteristic cell of these nodules is found to be a large giant cell closely resembling a neuroblast. Nests of atypical glial cells may be found. There are defects in the arrangement of the cortical layers of cells.*

**Klippel Feil Deformity** This peculiar condition is distinguished by a congenital absence of the upper cervical vertebrae so that the neck is extraordinarily short or may even be absent altogether. The lowering of the hair line on the back of the neck and the limitation of motion are also characterising features. The malady is not progressive and does not shorten life. No treatment is of any benefit and the importance of recognising the condition rests on the fact that these children may be operated upon or treated under the mistaken diagnosis of congenital wry neck or Pott's disease.

**Mental Deficiency in Infancy** Recognition of mental deficiency at birth or during early infancy is not easy unless there are present gross physical defects such as hydrocephalus diplegia and Mongolism. So little is expected of the infant during these early months that it is only when more complex acts such as walking and talking are slow in developing or fail to appear that the parents realise that something is wrong. Too frequently one is assured that the child at birth was perfectly normal but that some adventitious circumstance

in the form of a fall or an acute infection appeared later and is to be blamed for the delay in normal development. It is essential therefore that the physician be cognizant of the various steps in the progress of development during the first two years of life. In the case of the first child particularly the parents may fail to recognise that something is wrong and there are plenty of well wishing persons ready to assure them that in time all will be well. This attitude may be sustained for a considerable time but sooner or later realisation of the true state of affairs will take place and any previous undue optimism on the part of the medical practitioner will *not* redound to his credit. Careful inquiry into the process of development in these first few months of life will reveal sufficient at least to make a guarded prognosis the wisest one.

At birth the infant should suck vigorously and problems in *suckling* are rarely encountered in normal children. As early as the third week the baby's eyes fix on bright objects and the head is turned towards the light. It is not however much before the third month that *conjugate movements* of the eyes are perfected and in default squinting may occur. By the fourth month moving objects should be followed smoothly and accurately and the infant should show a tendency to *reach for objects* with his hands. From the fourth to the sixth month a child should *recognise first his mother* or nurse and later his father as well as other members of the family. *Awareness to strangers* occurs towards the ninth month. *Pictures* are recognised between the eighteenth and thirty sixth month of age.

The aimless movements of the infant gradually become transformed into co-ordinate activity. Attempts to *hold up the head* are made at about the eighth week and should be completed between the fourth and sixth month. At four months the baby *kicks and splashes in his bath* and should be starting to crawl on the floor. At the sixth month he should be able to *sit up alone* and by the ninth he should be able to pull himself up and stand with support. From the ninth to the twelfth month with help he is able to *walk*. Walking alone varies considerably with different children but is usually accomplished between the twelfth and eighteenth month. During the same period the child is able to climb stairs.

The development of *speech* is of great importance in assessing

the mental development of the child, inasmuch as it represents a more recently acquired faculty of human intelligence. The earliest expression of the infant's emotions is in the form of crying. Hunger, pain, anger, and fear—primitive instincts—find their expression in the infantile wails during the first few months of life. By the fourth month there is cooing and babbling, and from the sixth to the ninth month the baby makes imitative noises such as ma ma, bye bye, and da da. *Spontaneous talking* should begin from twelve to eighteen months of age. *Sentences* begin to appear thereafter or up to the second year of life. Before the third year the child should be able to ask questions. At the end of the first year of life the child is able to control the sphincters under reasonable conditions by day, but difficulties by night continue until the end of the second year or later. By fourteen months the child has some consciousness of the problem involved, and often is able to make known his needs.

### Normal Development

MONTHS	ACCOMPLISHMENTS
1 to 3	Fixes eyes on bright objects Turns head towards light Attempts to hold up head Sucks vigorously at breast or bottle
3 to 6	Shows primitive instincts by means of wails Eyes make accurate conjugate movements in following objects Reaches for objects Recognises members of family Holds up head Kicks and splashes in bath Crawls energetically on floor Coos, laughs and babbles
6 to 9	Is aware of strangers Sits up alone Makes imitative noises, as ma ma da da, bow wow wow, moo moo, etc
9 to 12	Pulls himself up and stands with support Walks with help Adds many new words to vocabulary
12 to 18	Controls sphincters in daytime and is able to communicate his needs Climbs stairs Walks alone Talks spontaneously
18 to 21	Begins to recognise pictures Speaks in sentences

21 to 24	Controls sphincters at night by 24th month
24 to 36	Repeats oft heard stories
	Asks intelligent questions
	Invents stories with himself as hero

Since the earliest development of the infant is in the sphere of muscular co ordination and the use of his special senses failure along these lines can be early appreciated. The child may be of the restless irritable variety screaming and wailing continuously and refusing to do anything in the first few months of life or apathetic and inert indifferent to his surroundings and making no attempt to help itself. With both these types the earliest sign of difficulty is the refusal to take the breast. Feeding problems are paramount suggesting that the biological interpretation might be that an attempt is being made to eliminate a defective organism. Later on in any given instance application of the rules of normal development given above will demonstrate the presence of feeble mindedness and its degree. Careful inquiry taking month by month the baby's development provided that the mother is sufficiently intelligent to co operate will make possible an early diagnosis and prognosis. The hardest problem is that of the deaf mute. However good muscular co ordination interest in the surroundings and cleanly habits will help to differentiate it from amentia. Recurring convulsions without obvious cause are so common in mentally deficient babies that their presence suggests some degree of mental impairment or at least raises grave suspicions of its appearance at a future date.

### Cerebral Palsies of Childhood Cerebral Diplegia Infantile Hemiplegia

The group connoted by the above title is a large one and the causes producing it are multiple. Three main causes have to be considered.

(1) *Damage to the foetal brain in utero*. This may be the result of infections and intoxications affecting the foetal brain and causing cerebral maldevelopment. Eclampsia influenza and other infectious diseases in the mother or exhaustion worry, and injury to the maternal parent may be factors. In a small proportion of cases heredity undoubtedly plays a part. In the rest there is no clue to be obtained from the history of the pregnancy.

(2) *Injury during birth* may occur by precipitate labour. Prolonged pressure of the foetal head during birth or extraction by forceps may play a part. While accepting these causes as possibilities the burden of proof rests on the assumption that the child was normal before birth.

(3) *Infection or Intoxication*. Occasionally new born children may be affected by extrinsic infections such as influenza, respiratory diseases and gastro intestinal maladies which in turn produce complications in the central nervous system. The intrinsic toxic encephalitis of infancy is however a much more common cause. This is abrupt in onset, short lived in duration and yet far reaching in its ultimate results. The infant may have a normal birth and delivery and a peaceful first few months of life when with catastrophic suddenness appears a high temperature associated with repeated convulsions, paralyzes and finally a cessation of all normal physical and mental development. Often these initial episodes are missed and the mechanism of birth is blamed for the later neurological condition.

Congenital syphilis is a very rare cause of cerebral diplegia.

To sum up, the larger proportion of the cerebral palsies of childhood are caused by intra uterine troubles producing the maldevelopment of the brain called agenesia. The next in importance are the infections and intoxications of early infancy. The last and the least important are the factors of birth injury.

*Diagnosis*. The recognition of the existence of cerebral palsy in an infant depends altogether on the retardation of the stages of development outlined above. Added to this are the factors of *spasticity* or *atonicity* of the limbs or involuntary movements of an abnormal character. Commonly the child fails to hold up his head or sit up at the proper time, the musculature is unusually rigid and there may be continuous bizarre spontaneous movements of all four extremities and face. *Mental deficiency* may be a prominent symptom or may be only associated with the muscular disturbance. It may range through all degrees from extreme idiocy to slight backwardness. Occasionally the muscular impairment is more marked than the mental defect and these children may show a relatively high degree of intelligence in later life.

The *lower limbs* are generally more affected than the upper and in the classical type of *Little's disease* there is only involvement of locomotion. In this condition the child when learning

to walk, is impeded by the *spasm of both adductor muscles* of the thighs so that the knees rub together or cross each other, producing the so called '*scissors gait*'. Involvement of the upper extremities, face, jaws and tongue, together with difficulty in breathing and suckling represent only relatively increased degrees of severity. Difficulties in enunciation, deglutition, and the dribbling of saliva are common in the more severe cases where the bulbar mechanism is involved. All tendon reflexes are increased and the condition is commonly symmetrical. The usual complaint, in a moderately severe case, is that at about six months of age the mother notices that the child is unusually rigid in the bath and does not make the full use of his limbs. The parents recognise that the condition is not progressive and that as time goes on, slow improvement may occur.

Spontaneous abnormal movements or *athetosis* as they are termed, frequently take place in these diplegic children. They are characterised by slow writhing twisting movements of all four extremities and are associated with grotesque facial grimaces. When the child is excited by tickling, fear, or laughter, the movements become more rapid and violent and shake the whole body. Along with athetosis there is a marked overflow of all muscular activity so that attempts to move even one little finger may bring the whole body into a rapid play of violent movement.

Since the aetiological factors are so variable the *pathological changes* in the brain are equally so. The common findings are

- (1) An unusually small brain
- (2) Hardening and atrophy of the cortex so that the sulci are widened
- (3) Simplification of the cortical pattern and absence of gyri normally present
- (4) Porencephaly or large defects in the cerebral hemispheres e.g., a tunnel may exist bringing the ventricle into communication with the outside of the brain
- (5) Microscopically there is an overgrowth of neuroglia, poverty of the layers of cells in the cortex, and various other malformations or deficiencies in the architecture of the cortex and white matter

Treatment of these cases of cerebral diplegia consists in making use of and encouraging the faculties still left to the child. In severe cases where the child is practically helpless,

or where the athetoid movements are universal and disabling, practically nothing can be done. In the milder cases where intelligence is preserved—fits are absent and the child has some use of its functions—a great deal can be done by *muscle training*. These children try to help themselves, show a certain amount of ambition from the start, and can be greatly improved by patient effort. The *Montessori method* of training of muscular co-ordination is undoubtedly of value.

**Infantile Hemiplegia** In this disease, as in the case of the diplegias, there are a very large number of possible causes operating during the early months of life. The difference is that while ante-natal factors are dominant in the cerebral diplegias it is very rare for an infantile hemiplegia to be present at birth. Much more common is the appearance of a one-sided paralysis in an infant following such diseases as whooping cough, measles, scarlet fever, diphtheria, chicken pox, small pox, vaccinia, pneumonia, and all the other toxic and infectious diseases of infancy. Many cases occur without obvious cause, so that the hemiplegia seems to result from a primary brain infection.

The pathology is obscure and many different lesions have been described.

**Symptoms** The onset of the hemiplegia is usually very sudden, occurring during the course of some infectious disease, and appearing about the second week of the illness, or sometimes not until convalescence has been established. Often there are convulsive manifestations chiefly on the side involved and with each bout of seizures there is increasing weakness of the arm, leg and face. Often there is high fever and coma during which an increased flaccidity may be noticed in the paralysed side. This becomes more obvious during recovery of consciousness and complete paralysis on one side of the body is apparent after a few hours of illness in a previously normal infant or in one recovering from a mild infection. In less severe cases a slight weakness of the arm or leg may appear suddenly and mysteriously without any fever, convulsions, loss of consciousness, or other apparent cause.

Recovery commences in a few weeks after the illness, but may take months to reach any degree of restitution of function.

The prognosis in a given case depends largely on the severity of onset, the continuance of convulsive movements, and the degree of one-sided paralysis. The convulsive seizures may

persist indefinitely in after life. A few weeks after the paralysis has been established the flaccidity becomes spasticity and contractures often take place. When the paralysis is in complete choreic and athetoid movements appear in later months and unfortunately these add to the disability. In any given case a definite prognosis should not be given for many weeks after the onset for there is a wide range of possibilities between complete recovery, partial disability and the unhappy triad of mental deficiency, paralysis and epilepsy.

Treatment during the acute stage resolves itself into

(1) Frequent lumbar punctures and when the spinal fluid pressure is increased as it usually is these may have to be done three times a day until the pressure becomes normal.

(2) Subcutaneous administration of sodium phenylbarbitone in doses of gr  $\frac{1}{2}$ -1 in a 20 per cent solution to control the convulsions.

(3) Glucose and saline should be given by rectum as long as there is coma.

(4) Later the resulting paralysis should be treated by massage and passive movements to prevent contractures.

**Encephalitis of Infancy** The vulnerability of the infantile brain has already been mentioned. There are so many toxic or infectious influences that may affect a young and developing brain that a classification based on exact pathological findings is impossible. The acute toxic encephalitis in childhood of Grinkler and Stone and the Strumpell Leichtenstern type of polio encephalitis are two of the very many varying types of inflammation of the brain in infancy. In some cases the main brunt of the damage is on the cellular components of the brain.

In other cases the white matter is the most affected and areas of demyelisation occur. In other instances the local vascular system is the most affected and endarteritis leading to obliteration of the lumen of the vessel occurs extensively. Perivascular infiltration so common in epidemic encephalitis and polio-encephalitis is not so common in these forms of brain infection.

It is not to be wondered at therefore that while encephalitis in infancy is so common the classification of these infections of the brain on a definite clear cut pathologic basis is next to impossible.

**Symptoms** Clinically these varieties of pathological process produce more or less similar effects. The onset is ordinarily

acute and may be fulminating. There may be an antecedent exanthema or pyogenic infection in which case the encephalitis appears within the second or third week of illness. In many other instances the signs of encephalitis are of spontaneous origin. Altogether the clinical picture is very similar to that already described as occurring in the course of infantile hemiplegia. Fever, convulsions, headache, vomiting, delirium and later coma are the rule. Monoplegia, hemiplegia and diplegia may occur and there may be signs of meningeal irritation in the form of rigidity of the neck. König's and Brudzinski's signs. The tendon reflexes may be diminished or lost. Involuntary urination and defecation may take place especially if consciousness is lost. Cranial nerve palsies are uncommon but blindness as a result of damage to the optic nerve or to the cortical centre is frequent. Paralysis may involve one side of the body or both lower extremities and the sensory changes are variable. The cerebrospinal fluid is usually under increased pressure and contains an abnormal amount of protein but there is little or no increase in its cellular elements.

The differential diagnosis involves exclusion of tuberculous or meningococcal meningitis. Here the spinal fluid examination settles the problem (see pp. 172-3). The prognosis in these cases varies from one epidemic to another but it is generally better than might be expected.

Treatment is by repeated lumbar puncture and the intravenous administration of hypertonic glucose solution. In the cases of encephalitis following acute infectious illnesses of infancy including vaccinia the administration of convalescent serum has been sometimes beneficial. Occasionally good results are claimed for the intramuscular injection of 10 c.c. of citrated whole blood. Lastly, hexamine may be given by mouth in doses appropriate to the age of the baby.

### Diseases Affecting the Cerebrospinal Vascular System

**Arterial Disease Secondary to Infections.** Rupture of a vessel, thrombosis and embolic occlusion may occur during the course of or following any of the infectious diseases of childhood. The clinical picture is usually that of a hemiplegia with convulsions and the onset is usually sudden. Much more rare is the appearance of a spontaneous vascular occlusion in an

infant wherein no antecedent history of illness could be obtained

The *etiological factors* involved here come under the three headings of *injury septic infection* and *marasmus*. The traumatic origin in infants is not common. It occurs frequently enough following transfusion of the infant by way of the superior longitudinal sinus to contra indicate this route for intravenous therapy. In septic infections of the ear and mastoid the *lateral sinus* may be involved.

**Symptoms** In such cases there is a high swinging temperature. Some venous congestion may be seen in the neighbourhood of the mastoid process and there is tenderness in the neck along the course of the jugular vein. Occasionally the vein can be palpated as a hard tender cord. Spinal puncture usually shows an increase in leucocytes both polymorpho nuclear and mononuclear indicating a localised meningitis. Compression of the jugular vein on the normal side will cause an increase in pressure in the spinal fluid as measured by a manometer. No such rise in pressure occurs when the neck on the side of the thrombosed sinus is compressed.

*Septic infections* of the anterior air sinuses such as the *sphenoidal ethmoidal and frontal* are more likely to involve the *caavernous venous sinus*. Symptoms of this complication are usually very definite. Besides the constitutional reaction to sepsis there is a marked oedema of the root of the nose and the eyelids and a conspicuous proptosis of one or both eyes. There are usually associated paralyses of the ocular muscles.

*Thrombosis of the superior longitudinal sinus* is seen more frequently in debilitated emaciated and marantic infants. The clinical picture is not always easily recognisable and often the thrombosis is found at post mortem having gone unrecognised during life. The general symptoms are headache vomiting convulsions and often retraction of the head. Thus it may simulate an oncoming meningitis. More localising in character is the tense fontanelle the marked congestion of the veins of the scalp and in some cases those at the base of the nose. Ocular squints may occur and papilloedema is often present. There may be a hemiplegia from obstruction of cortical veins entering the sinus on one side. Convulsive movements of both lower extremities followed by a paraplegia are rarer yet very characteristic signs of the condition. In severe

cases all four extremities are paralysed shortly before death. Except in the case of lateral sinus thrombosis following middle ear disease the *outlook* in all cases of thrombosis of the large intracranial venous sinuses is bad. The mortality is very high and *treatment unavailing*.

**Amaurotic Family Idiocy** This is a hereditary disease affecting chiefly the Jewish race, particularly in the infantile form. As the name implies, the two cardinal features are *blindness* and *idiocy*. At birth the child is normal and development proceeds without interruption until between the fourth and sixth months of life. The child then ceases to take notice of its surroundings and any acquired traits such as holding up the head are rapidly lost. The parents notice that the baby has become blind and all four limbs rigid. At this stage the arms are extended and rotated inwards, while the



FIG. 27.—Amaurotic Family Idiocy. Advanced Stage of Disease. (With kind permission of Dr. J. I. Steen.)

legs are also extended adducted and crossed. A characteristic feature is the child's reaction to a sudden noise like a loud handclap. Following this stimulus there is a marked generalised spasmodic contraction of all the muscles of the body. Later on the muscular rigidity is replaced by universal flaccidity, and the child dies a few months after the onset.

**Diagnosis** Recognition of the disease rests on the characteristic findings of *ophthalmoscopic examination* the *progress of events* and the fact that the child is of *Jewish race*. The optic discs are pale and show evident signs of atrophy and in the macular region of the retina there is a *cherry red spot* surrounded by a halo of lighter colour. In a well developed case the obvious idiocy blindness and emaciation are sufficient to make the diagnosis.

There is no *treatment* of any value.

*The pathological processes* involved are essentially those of a swelling and lipoidal degeneration of the ganglionic cells of the cortex thalamus cerebellum and spinal cord. There is a disturbance of lipoid metabolism due probably to endogenous intoxication. There are certain factors which seem to connect this disease with other familial lipoidal metabolic disturbances such as Niemann Pick's disease *e.g.* the enlargement of the spleen and liver occurring early in infancy the Jewish parentage etc.

As a contrast to amaurotic family idiocy wherein the chief damage is to the ganglionic cells there is also a poorly defined group of progressive familial disorders of infancy wherein the brunt of the assault is borne by the subcortical white matter. In these cases there is a widespread demyelination of the nerve fibre. Later and in chronic cases a marked neuroglial overgrowth occurs.

**Symptoms** The common picture which these cases present is that of a previously healthy child who develops during the first year of life paralysis convulsions mental deterioration and blindness. Optic atrophy or swelling of the optic discs may be present. The onset may be abrupt with fever headache and rigidity of the neck and the vomiting and papilloedema may suggest the presence of tumour. In other cases the onset is more gradual the disease is less active in its progress and ataxia tremor and slow speech may be prominent features. Essentially these diseases are familial and progressive but up to the present they have been poorly defined their syndromes being more variable than that of familial amaurotic idiocy. Included in this group are the encephalitis periaxialis diffusa of Schilder the familial infantile form of diffuse brain sclerosis of Krabbe and lastly the aplasia axialis extra corticalis of Pelizaeus and Urbachier.

**Intoxications and Deficiency Disorders** The influence of

*diphtheria* in producing damage to the peripheral nervous system is mentioned elsewhere and there are many other less obvious endogenous toxins affecting an infant. There is however less chance of the baby being affected by toxins outside the body since its environment is relatively well protected. In recent years the possibility of *lead* entering the system of young children has received considerable attention in the publications of McKhann and Vogt. It is possible that many puzzling cerebral symptoms of young children may be the result of *lead poisoning*. As a source of lead it is necessary to consider nipple shields worn by the mother, the face powder she habitually uses and the domestic water supply. The most common source of lead however in the case of young children is in the paint of toys and beds which may be chewed or sucked.

The earlier symptoms are gastro intestinal in character—*e.g.* abdominal pain, constipation and lack of appetite. Cerebral symptoms appear later and are characterised by vomiting, *papilloedema*, convulsions and stupor. The pulse rate may be slowed, the blood pressure may rise and *x ray* may show separation of the cranial sutures. The clinical picture therefore is of increasing cranial pressure due to *oedema* of the brain. The ordinary signs of lead poisoning in adults such as the lead line in the gums, changes in the blood and neuritis are seldom present.

The diagnosis is made usually by the history of ingestion of lead in some form or other and the characteristic *x ray* finding in the bones. This is in the form of a broad opaque band at the epiphyseal ends of the long bones. Treatment consists in the use of potassium iodide, large doses of calcium with *viosterol*, cod liver oil and exposure to sunlight.

*Tetanus neonatorum* fortunately is a rare disease. The entrance of the organism is through the umbilical wound. Symptoms appear between the second and fourteenth days and as in adults the earlier the onset and the more rapid the progress the more serious is the prognosis. Feeding is difficult because spasms of the jaw occur as soon as the child is put to the breast. The facial muscles go into a spasm and the *risus sardonicus* appears. There is an extreme irritability of the child to external stimuli, such as a bright light or a sudden noise. At first the muscles of the body go into contraction, later generalised convulsions appear. The outcome is very serious.

The treatment consists in large doses of anti tetanic serum given intramuscularly as much as 10 000 units should be given in the first twenty four hours. Intrathecal administration does more harm than good. For the rest it is a question of giving sufficient sedatives to control the spasms without killing the infant. Potassium bromide and chloral hydrate of each three grains given by rectum are of advantage and absolute quiet and preservation of the infant from external stimuli are essential. As in adult tetanus prophylaxis is more important than treatment indeed with modern aseptic methods this disease should not occur.

### Pink Disease Erythrœdema Polyneuritis Trophodermatoneurosis Vegetative Neurosis Acrodynia

These various complex names refer to a disease recently described and the variability in the nomenclature indicates our lack of knowledge as to the exact ætiology. Nevertheless the condition is a true clinical entity varies little from case to case and once seen can be easily recognised in future. The arguments as to whether it is a deficiency disorder or due to a virus infection cannot be discussed here. Perhaps it is best classified as a disease due to deficiency in some vital element.

Children are chiefly affected between the ages of four months and seven years. The commonest period is between nine and eighteen months. Male children predominate slightly. The disease commonly appears during the winter months and may occur in local epidemics. There is no evidence that it is contagious. It was early recognised that the clinical disturbances were chiefly in the vegetative nervous system.

**Symptoms** The commonest early symptom is irritation of either the respiratory or gastro intestinal tract in the form of bronchitis or diarrhoea with or without fever. Following these common afflictions of childhood there is a complete change in the whole aspect of the child. He becomes miserable whining irritable his sleep is disturbed and his appetite disappears. There is continuous crying temper tantrums occur and the child becomes increasingly difficult to comfort or feed. There is also an extreme susceptibility to light and the hands and feet become bluish red swollen glazed and cold. Because of these symptoms the unfortunate child adopts a *characteristic attitude*. *He buries his face in the pillow, tucks the hands and*

feet under his body for warmth, and elevates the buttocks. It is quite characteristic on entering the ward to see a miserable, whining salivating child crouching in this position. He resists vigorously any attempt at examination and fights to be left alone and to be allowed to return to his previous position. Sweating is marked and associated with this there is often a rash erythematous or papular in character. In ordinary cases desquamation occurs on the hands and feet and ulceration of the mouth falling out of the teeth nails and hair may occur. The muscles become hypotonic and the tendon reflexes disappear. The pulse is persistently fast (about 160 to 180) and



FIG. 28.—Erythrodema Polynarium or Pink Disease. The child adopts this attitude because of photophobia and painful extremities.

the blood pressure raised to 110 or 130 mg of mercury. The rash is intensely irritating and the child tends to bite its fingers. Insomnia may become acute.

Death as a result of cardiac failure or broncho pneumonia may take place. Fortunately the mortality is low (i.e. about 5 per cent). The majority survive but the disease is a protracted one and lasts from a few months to over a year.

Treatment is entirely symptomatic. Feeding by gavage is sometimes necessary. Most important of all the baby must be kept clean and dry. To combat the irritability small doses of luminal chloral or bromides are necessary and splints gloves or stockings are necessary to prevent scratching. Because the disease has been considered to be a deficiency

disorder, the use of 2 oz. of raw liver daily has been advised. Cod liver oil, viosterol, and ultra violet light are helpful. Transfusion may be very beneficial. Recently, treatment by placing the child in a room with windows of red glass has been recommended. It is doubtful if the benefit claimed in these cases is due to anything more specific than a relief of the photophobia, which may be very severe and lead to great restlessness if the child is left in a bright light—but the essential thing is to make the child as comfortable as possible while awaiting the usual spontaneous ending of the disease.

*Poliomyelitis.* Readers are referred to the section on Communicable Diseases and text books on general medicine.

## CHAPTER XXVI

H L PARKER AND DOROTHY PRICE

### DEFECTS AND DISEASES OF MUSCLES

(*Congenital Defects—Congenital Absence of the Abdominal Muscles—Congenital Torticollis—Diseases of the Muscles—The Pseudo-hypertrophic Type—The Juvenile Type of Erb—The Facio-scapulo-humeral of Landouzy Dejerine—Amyoplasia Congenita—The Familial Muscular Atrophies (Amyotonia of Oppenheim and Progressive Spinal Muscular Atrophy of Werdnig Hoffmann)—Myotonia Congenita*)

**Congenital Defects** Congenital defect or absence of certain muscles is not uncommon. A whole muscle or a portion of it may be absent or the muscle represented merely by fibrous tissue. The muscles most commonly affected are the pectorals, trapezius, serratus magnus and quadriceps. Congenital bilateral ptosis has been described as not uncommon in America. Often the defect gives rise to no abnormality.

*Congenital absence of the abdominal muscles* is an interesting and rare condition. The abdominal wall is very thin in this condition and the skin over the abdomen has a characteristic wrinkled grey appearance. The defect gives rise from birth to respiratory difficulty, the baby being unable to cry or cough. Usually, death occurs from the first respiratory infection. Sometimes the condition is associated with malformation of the ureters and bladder musculature.

*Congenital torticollis* or wry neck is a condition in which the muscles of the neck are shorter on one side than on the other at birth. Its cause is obscure. Contracture following hæmatoma of the sternomastoid due to birth injury occasionally may be cited as the cause, but in severe cases all the tissues on one side of the neck show shortening and it must be supposed that the condition has arisen *in utero* due to faulty position of the foetal head. In the worst cases the muscle may be replaced completely by fibrous tissue.

Treatment consists in manipulation or open operation (see p. 394).

**Diseases of Muscles** Many of the diseases of muscles are rarely if ever seen during infancy, hence only the commoner

varieties and those which have been reported from time to time as starting during the first twelve months of life are mentioned here

*The Myopathies* (or muscular dystrophies) are due to primary wasting of the muscles unassociated with nerve lesion though secondary degeneration of nerve elements may follow the condition. They are familial and occur more commonly in boys than girls may be present at birth or appear during infancy and are steadily progressive. Several types have been described e.g. the pseudo hypertrophic juvenile facio scapulo humeral and distal (Wylie)—but the essential lesion is fundamentally similar in all. The muscle fibres waste and finally disappear. In this process of degeneration certain fibres may appear temporarily to hypertrophy to several times their normal size but gradually all the fibres become wasted and replaced by fibrous tissue. At the same time fat may be laid down between them so that the total bulk of the muscle appears greater than normal. In the end however this fat also disappears and only contracted fibrous bands remain. The clinical appearances of the different types will depend upon the relative amounts of fibrous tissue muscle tissue and fat present at any one time.

*The pseudo hypertrophic type* is the commonest variety it rarely affects the female but may be transmitted through the mother. The onset is seldom before the second year most commonly between the fourth and sixth. The first symptoms for which the child is brought to the doctor is usually weakness of the legs associated with apparent enlargement of the muscles of the calves thighs and buttocks. Later the shoulder girdle and arms become involved. These children are apt to topple over if left unsupported. They show a most characteristic method of rising from the floor—they climb up themselves—e.g. the child turns his face downwards and raises himself on his arms and legs then he throws his weight solely on to the legs supporting it partly through his hands which he first places on his knees and then moves upwards hand over hand till the trunk is fully upright when he stands unsteadily with a marked lumbar lordosis the shoulders hunched back and the scapula projecting. At first the reflexes are normal but gradually they become diminished as the disease progresses as also does the muscle response to galvanism and faradism though the reaction of degeneration is never shown.

The progress of the disease is steady, the end coming in five to ten years through intercurrent infection, no treatment being of any avail

*The juvenile type of Erb and the distal type of Gowers* do not commence till after the tenth year and hence need no description here

*The facio scapulo humeral type of Landouzy Dejerine* is characterised by weakness of the muscles of the face and 'winging' of the scapulae. It may be present at birth, in which case the first symptom will be failure to suck at the breast or bottle. At the same time it may be noticed that the baby lies with his eyes half open during sleep. As the child grows the weakness of the muscles of the face becomes more marked and lack of facial expression becomes obvious. The muscles of the eyeball tongue pharynx and larynx are unaffected. The condition may remain stationary for many years or the shoulder girdle may be affected during early childhood. Later the pelvic girdle tends also to become affected.



FIG. 23.—Amyoplasia Congenita. Baby aged 4½ months showing characteristic appearance of arms

**Amyoplasia Congenita (Arthrogryposis Multiplex Congenita)**  
This rare condition may be defined as an immobility of one or more limbs. The rigidity occurs usually bilaterally in the elbows or knees, which are fixed in extension. The condition appears to affect a whole group of muscles, either extensors or flexors hence, by the unopposed action of the opponents, contraction or rigid extension ensues. The spine itself is never affected.

The condition was formerly considered to be a disease of the joints, and these cases usually came into the hands of the

orthopedic surgeons. Recent work \*, † however, goes to show that the pathology consists essentially in a degeneration and fat replacement of the muscles, probably due to an intra uterine arrest of development.

The condition is found at birth and treatment consists in obtaining movement in the affected joints by massage and passive movements and by surgical orthopedic measures to correct gross deformity.

**The Familial Muscular Atrophies of Infancy** There are two diseases that come under this title *Progressive spinal muscular atrophy* originally described by Werdnig Hoffmann and the *amyotonia congenita* of Oppenheim. The task of describing these diseases has not been made any easier by observers who claim



FIG. 30.—Amyoplasia Congenita. Baby aged 4½ months showing characteristic appearance of arms.

that there are transitional forms blending one into the other. Actually it has been suggested that there is no distinct difference. For practical purposes however it is better to take the extreme forms of these two diseases and retain their separate identities.

*Amyotonia congenita* is occasionally but not often familial. That the disease is present at birth is borne out by the fact that the mother notices the extreme flaccidity of the infant immediately after birth. Its limbs can be moulded into grotesque forms and there is much delay in holding up the head and sitting up. Movements are feeble and sometimes the child assumes strange postures because of the atony of the muscles. For example on being placed in the sitting up position the trunk may fall forward and lie between the

\* Sheldon (*Arch Dis Child*, 1932, VII, 117)

† Price (*Arch Dis Child*, 1933, VIII, 117)

abducted lower extremities, and on attempting to raise the child, the head if unsupported may fall backward at an alarming angle. When the child is placed on a hard surface the muscles seem to "flow" evenly as if they were of a liquid nature. The outstanding feature is that there is nearly always preservation of the facial and bulbar musculature, for which reason survival is possible. The deep reflexes are diminished or abolished, there is no marked atrophy, and the baby presents a normal intellectual development. Improvement in these cases may occur, and although the majority die from intercurrent affections, a few recover and mature, in spite of a retarded development of muscular activity. There is never complete recovery,



FIG. 31.—Werdnig-Hoffmann's Disease. Baby, aged 4 months, showing muscular wasting and paralysis of the shoulder girdle and intercostal muscles

and a good many of the *professional contortionists* of adult life are cases of this disease.

The *pathology* is mainly muscular in character, but there is a relative diminution of the anterior horn cells of the cord. The muscle cells are very small and are largely of the foetal type.

*Werdnig-Hoffmann's disease* has a much graver prognosis, and in a well-developed example of the disease the course is ruthlessly progressive from the start. It is *not* congenital in origin, but appears at any time during the first year of life. The infant is apparently normal at birth, but sometime during the first few weeks or months of existence paralysis appears in the muscles of the shoulder and pelvic girdles. Movements already learned are lost. Loss of power is first noticed in the muscles operating around the hip joint, then the back muscles, so that a child who could sit up is now unable to do so. Later

on the muscles around the shoulder are affected and at this stage of the disease the characteristic appearance may be that of a child totally unable to move his hips trunk or shoulders while still retaining the normal use of his hands and feet. The neck muscles ultimately become affected and paralysis of the bulbar mechanism of swallowing usually closes the picture. The diaphragm always remains unaffected. The paralysed muscles undergo atrophy later and fibrillary twitching may be seen although the subcutaneous fat may cloud this important sign of lower motor neuron disease. In both conditions the action of the sphincters remains unimpaired. The deep reflexes are abolished early but there is not the same degree of universal flaccidity of the muscles as in Oppenheim's disease.

The pathologic changes show a much more marked degeneration of the anterior horn cells in the progressive spinal muscular atrophy of Werdnig Hoffmann and moreover the degeneration is a more active process and not a mere absence of anterior horn cells as is seen in Oppenheim's disease.

The differential diagnosis of these muscular atrophies from other conditions depends largely on the familial history. This may be absent or the baby may be a first child.

*Diphtheritic polyneuritis* presents few difficulties because of the obvious history of exposure to and clinical evidence of diphtheria. Palatal weakness is usually present and the muscular paralysis is widespread. Further the bulbar symptoms are in reverse order in that the swallowing difficulty is early and the muscular weakness later in appearance. Rickets may produce a similar picture of muscular flaccidity but skeletal changes as shown by x ray will help to differentiate. Following any debilitating illness there may be weakness and atony of the muscles but the history is suggestive and recovery is prompt. The greatest difficulty in the diagnosis of Oppenheim's or Werdnig Hoffmann's disease rests in the exclusion of the more common group of atonic types of cerebral palsy of childhood. In early infancy the clinical differentiation is impossible. Later the idiocy of the child shows that one is dealing with the flaccid type of cerebral palsy or the so called cerebro cerebellar diplegia wherein amentia is the rule.

Treatment is unavailing in Werdnig Hoffmann's disease. The condition proceeds ruthlessly to a fatal termination in child after child born of a family thus tainted. In Oppenheim's disease careful feeding supportive measures protection from

infections and light massage may bring the infant to the survival period and ultimate maturity. Unhappily this can seldom be accomplished.

*Myotonia Congenita*. This is a familial disease first described by Thomsen and sometimes called by his name. It occurs in both boys and girls and may commence in infancy, childhood or adult life. It is characterised by a delay in the response of the muscles to stimulation and by their failure to relax immediately when contraction is over. The condition is best demonstrated if the muscles of limbs are called upon for a sudden movement. For a few seconds the patient remains motionless then the muscle contraction takes place and is followed by a slow relaxation. If the movement is repeated several times "the latent period" becomes less marked.

Difficulty in sucking is the earliest recorded symptom. Meara described a case which on commencing to walk did so for the first few paces on tiptoe.

Section of the muscles only shows decreased striation and some enlargement of the muscle fibres.

The disease lasts throughout life and treatment is unavailing.

## SECTION VI

P MACCARVILL

### CHAPTER XXVII

#### DISEASES OF THE SKIN

(Introduction—Congenital Affections of the Skin Ichthyosis, Albinism Nævi—Eruptions Chiefly Due to Local Causes Eczema Napkin Erythema Intertrigo Sudamina—Eruptions Due to Animal Parasites Scabies Pediculosis Fleas—Eruptions Infective in Origin Streptococcal Infections (Bullous Impetigo Dermatitis Exfoliativa Neonatorum Common Impetigo Contagiosa etc) Staphylococcal Infections (Impetigo of Bockhart Multiple Subcutaneous Abscesses in Infants Seborrhœic Dermatitis Molluscum Contagiosum Thrush Congenital Syphilitic Lesions, Herpes Eruptions of Toxic Origin Exanthemata—Eruptions Due to Food Toxins Sera Enema Rashes etc Vaccination Rashes Drug Rashes—Care of Normal Skin Appendix of Useful Prescriptions.)

#### Introduction

THERE are certain diseases of the skin which are peculiar to infancy others are more prevalent in infants than in adults and many present characters modified to such an extent in infancy that they show altogether different features from the same diseases in adults Impetigo is essentially a disease of infancy and childhood though not confined to that age Nævi and other congenital conditions come under observation first at that time of life The eruptions of eczema and scabies have special features in infancy At this age the toxic erythemata are liable to be confused with the rashes of the specific fevers though the latter are rare in the first year In the following pages the common skin diseases seen in early infancy are briefly dealt with the rarer diseases being left to text books of dermatology In the treatment only measures capable of being carried out by the clinician will be detailed As radium and x rays can only be handled by those skilled in their use no reference to the technique will be made

#### CONGENITAL AFFECTIONS OF THE SKIN

##### Ichthyosis

Synonyms Fish skin Xeroderma Ichthyosis simplex

*Ichthyosis simplex* or *foetalis mitior* (ordinary type) *Ichthyosis foetalis gravior* (harlequin foetus)

**Definition** An abnormality of congenital origin appearing at birth or during the first year and characterised by roughness, scaling and dryness of the skin

**Ætiology** The cause is unknown but it has been suggested that it is due to some defect in the thyroid secretion. The disease is often hereditary

**Signs and Symptoms** All degrees of the disease occur from a slight dryness with branny scaling up to the condition described as "harlequin foetus" in which the child is born prematurely and appears to be encased in an armour of horny plates. Such infants are either still born or only survive a few days. In the slight cases (*xeroderma*) there is dryness and slight scaling. On the extensor surfaces of the limbs and on the buttocks there may be hyperkeratosis around the hair follicles causing a 'goose skin' appearance.

In the more severe cases there is a heaping up of greyish brown scales especially on the extensor surfaces. The flexures of the large joints are usually normal. The hair is dry and scanty, the horny layer of the palms and soles is thick and may be cracked, while the nails may be thickened, ridged and opaque. The mucous membranes are not affected.

The ichthyotic skin is particularly liable to eczema and to dermatitis from external irritants.

The skin is difficult to keep clean and too much attention in the form of cleansing agents, such as soap leads to soreness and cracking.



FIG. 32.—Severe case of ichthyosis. Flexures fairly normal

**Diagnosis** The presence of the condition at this early age the general dryness of the extensor surfaces with normal flexures makes the diagnosis simple

**Prognosis** The disease is likely to increase up to puberty and then remain stationary. It is not curable but treatment will ameliorate it.

**Treatment** *Internal* Thyroid gland extract is the only internal treatment of any value. Its action is variable and improvement only occurs while it is being taken. The dose should be pushed until improvement takes place or signs of hyperthyroidism appear.

*External* This is the most important part of the treatment. A daily warm bath should be given to which has been added starch or bran or bicarbonate of soda (one teaspoonful to the gallon of water) and the use of a superfatted soap. After drying the skin should be lubricated with salicylic acid 2 per cent in equal parts of olive oil and lanoline. The baby should be warmly clad and not exposed to harsh winds.

### Albinism

**Definition** A congenital absence of pigment from the skin hair and eyes.

**Ætiology** Cause unknown. It is occasionally hereditary and familial.

**Signs and Symptoms** Albinism is usually complete partial cases being very rare. When it does occur the pigment free areas are arranged in a segmental or zoniform manner (nævus achromicus). The albinos have white skin pink irises and white or flaxen-coloured hair. Having no pigment in the choroid they suffer from photophobia and nystagmus. For want of its protecting pigment the skin reddens and blisters easily when exposed to the sun.

**Prognosis and Treatment** The condition is permanent. No treatment is effective. Protection of the skin from the sun and shading of the eyes by tinted glasses will be necessary.

### Nævi

**Synonym** Birth marks. Moles.

**Definition** Nævi are localised abnormalities of congenital origin usually hyperplasias of some elements of the skin or

subcutaneous tissue Very rarely a nævus is a hypoplasia, e.g., nævus anæmicus, nævus achromicus

**Ætiology** Unknown They are often hereditary They frequently occur on sites corresponding to embryonic clefts, and are often associated with other developmental abnormalities, such as hare lip, webbed fingers, spina bifida, etc

**Classification of the Commoner Forms** (After Roxburgh)

Vascular nævi consisting of blood vessels	A Capillary nævus, nævus flammeus (port wine stain)	
	B Cavernous nævus (strawberry mark)	
	C Stellate nævus or nævus araneus (spider nævus)	
Vascular nævus consisting of lymphatic vessels	Lymphangioma circumscriptum	
Non-vascular nævi	(1) Plane	Pigmented macules
	(2) Raised	A Soft nævi, moles non-pigmented Pigmented Hairy Giant
		B Hard nævi Verrucose Linear, segmental Ichthyosis hystrix
		C Fibromatous Neurofibromata Von Recklinhausen's disease

### Blood-vascular Nævi

#### *Capillary nævus*

**Definition** A localised area of the skin in which the superficial capillary vessels are dilated and more numerous than usual giving the skin a pink, red or purple colour In its typical form the lesion is not raised though on its surface sessile cavernous angiomata frequently develop

**Clinical Features** Usually situated on the face and very common on the occipital region where it is usually pink in colour In size it varies from  $\frac{1}{4}$  inch to extensive areas It is usually noticed at birth or a few days later

**Prognosis** If present at birth and pink in colour it may disappear in a few months. If it persists after the first few months it will remain permanently and will increase in proportion to the growth of the child. (A B—This is the only type of birthmark in which treatment should be delayed in the hope of spontaneous cure. Cavernous angiomas sometimes disappear spontaneously but will more often increase fairly rapidly in size making satisfactory treatment more difficult.)

**Treatment** Is generally unsatisfactory. Freezing with carbon dioxide snow does not give satisfactory results and the same applies to the use of radium and electrolysis. Ultra violet light either from an air-cooled mercury vapour lamp or applied with pressure from a Finsen or Kromayer light gives the best results. In either case the dose must be such as to produce blistering and the treatment repeated when the reaction has subsided. The treatment may have to be continued for several months but in the majority of cases distinct improvement in the colour will be obtained and in the pink and red forms it will almost disappear.

### *Cavernous Nævus*

**Definition** A localised area of skin in which the capillaries are dilated and widened out to form hollow spaces. The walls of the vessels are thickened.

**Clinical Features** The lesions are usually raised and may be partly subcutaneous. This type has a red patch of dilated vessels on the surface, a surrounding area bluish in colour where the subcutaneous vessels show through the skin and further out usually an area covered by normal skin raised above the surface through which the underlying nævus can be felt. The nævi are red or purple in colour and vary in size from  $\frac{1}{2}$  inch to 2 or 3 inches in diameter. They are usually soft and compressible but may not be so if there is much fat and/or fibrous tissue present. They vary in prominence with the venous pressure (e.g. when situated on the face or neck they increase when the child cries). They may be present at birth but more commonly are not noticed for some weeks afterwards. They are commonest on the face and scalp but may occur anywhere on the body and are not infrequently multiple. They are frequently injured and bleed freely but the bleeding is readily checked by pressure. If broken they become septic and this

often causes disappearance of the naevus though usually incompletely the marginal portion being left

**Prognosis** They usually increase in size fairly rapidly, especially in loose tissue such as the eyelids and lobes of the ears They sometimes disappear towards the end of the second year but it is not advisable to wait for this event if the naevus



FIG. 73.—Cavernous naevus. A common site. (From Dr. V. S. Finzi's article in the *British Medical Journal* September 28th 1935 with permission.)

is at a site liable to rapid spread or if it is showing signs of enlargement

**Treatment** This may be by excision freezing with carbon dioxide snow electrolysis multiple puncture with galvanocautery or diathermic needle and radium. Excision is suitable for small naevi with definite edges where the skin can be brought together readily and on parts where scarring is of no importance. Carbon dioxide snow is only useful where the naevus is such that it can be frozen through completely. It is therefore of no use where there is subcutaneous involvement. The snow should be applied over the whole surface and a little outside the visible edge. The pressure should be firm and the application should last from 20-60 seconds. A few hours after

the application a blister forms and later a scab. After two to three weeks the scab falls off and improvement will continue for a further five to six weeks. If necessary the process can be repeated. If the case is suitable it has the advantage that it requires no anæsthetic.

*Electrolysis* If done with a single needle this requires several sittings and an anæsthetic each time. It will effect a cure in cases unsuitable for carbon dioxide snow. The procedure is best carried out however with a multipolar needle when much more can be accomplished at a single sitting.

*Multiple Punctures with Galvano-cautery or with Diathermic Needle* Treatment by either method must be carried out under a general anæsthetic and if the punctures are placed sufficiently widely apart to prevent scarring a quick and fairly good cosmetic result can be secured. They are most suitably employed for cavernous nævi affecting the mucous membrane surfaces.

*Radium* This is the method of choice but if it is going to be used it should be employed at once before other methods have produced scarring.

The gamma ray is generally used and it is applied either as a surface application or by the introduction of needles or screened seeds depending on the site and type of the nævus. The results are for the most part excellent especially if used in early infancy.

### *Stellate Nævus*

**Definition** A small type of nævus having a bright red central vessel about 1 mm in diameter from which radiate tiny capillary vessels giving the appearance of a spider.

**Clinical Features** It is not usually seen in early infancy, but is common in later childhood and adult life and is probably not a congenital defect. They are commonest on the nose and cheeks and have no tendency to disappear spontaneously.

**Treatment** Destruction of the central vessel by the galvano-cautery, carbon dioxide snow or electrolysis is followed by disappearance of the whole nævus.

### *Lymphangioma Circumscriptum*

This is a comparatively rare form of nævus consisting of a group of vesicles in the corium formed by dilatation of lymph

phatic vessels. They occur as a patch of white or grey translucent vesicles firm in consistency. They are commonest on the neck, upper limb and sides of the trunk, but sometimes occur on the tongue. They are best treated by excision.

### Non-vascular Nævi

(1) **Plane-pigmented Macules and Patches.** These are very common and frequently multiple. They are usually referred to as flat moles to distinguish them from the raised hairy type. They are so well known as not to merit description. They are best removed by electrolysis.

(2) **Raised Non-vascular Nævi.** A *Soft Nævus*. These all come under the common heading of "moles" and may be non-pigmented, non-hairy, hairy or giant. When hairy the hair occurs in tufts and is strong. In the giant there is a large area covered with deeply pigmented raised hairy patches. In rare cases these may cover the whole trunk or the bathing drawers area. None have any tendency to disappear spontaneously and some after middle life undergo malignant change. The smaller moles may be removed by electrolysis or carbon dioxide snow. The non-pigmented type occurring on the face can be pared down level with the skin, bleeding being stopped by pressure.

Large hairy moles occurring on the face can sometimes be excised, skin grafting then being performed on the denuded area. Moles which are not on the exposed surfaces of the body and are not subject to friction are best left alone.

B *Hard Nævi*. These are characterised by an overgrowth of the epidermis especially of the horny layer. There are no changes in the deeper layers of the skin and no nevus cells. They may be of all sizes from groups of small warty lesions a quarter of an inch in diameter, to horny patches covering the larger part of a limb. They comprise the *verruccose nævus*, which is a patch of rough warty skin on the trunk or limbs, the *linear nævus* which consists of streaks or bands of irregular width occurring usually on one side of the body only, *ichthyosis hystrix*, in which the lesions are horny, dark brown or black in colour projecting a quarter or half an inch above the surface.

The diagnosis of these types is not difficult, confusion only arising with plane warts, common warts or linear lichen planus.

The history however is usually sufficiently definite to enable a clear diagnosis to be made

In the milder cases treatment consists in keeping the lesions flattened by the use of a 2-10 per cent resorcin or salicylic acid ointment but in the larger and thicker lesions nothing short of excision followed by a skin graft is of any use

Neuro fibromata and Von Recklinhausen's disease are comparatively rare and will not be considered here

## ERUPTIONS CHIEFLY DUE TO LOCAL PHYSICAL CAUSES

**Eczema** The use of this term is gradually being narrowed by the exclusion of many eruptions hitherto called eczemas. There is as yet no general agreement as to the type of eruption to which the term should be limited. This is particularly so in the case of what are called 'infantile eczemas'. There occurs however in infants a skin eruption with constant well defined clinical features which most will agree to call eczema and to this type of catarrhal inflammation non bacterial in origin the term is here applied

**Ætiology** Various theories have been put forward as to the cause of eczema in infants. Digestive disturbances, dentition, vaccination, a diathesis, heredity, allergy or protein sensitisation all have their adherents.

It would appear that many of these factors if not actually causative aggravate the attack when present but it must also be admitted that to give sole attention to any one of them will not cure infantile eczema. The present writer agrees with Adamson that external irritants are probably the most important factor in the production of the disease with possibly an underlying diathesis which renders the skin of certain infants more susceptible than others to external irritants. The parts affected primarily are those subject to every external influence. The face exposed to sun and wind, changes in temperature, soap and hard water is almost invariably the site of origin and in those other cases where it appears to start in a scurfy scalp it can well be argued that the scurfiness is the irritant.

When once the condition has started it would appear that external irritation is the chief cause of its continuance and certainly the cause of spread. Against this one must admit that a certain number of infants suffering from eczema also

suffer from asthma which suggests some agent responsible for a hypersensitivity of mucous membranes and skin. The fact, however, that external measures alone can cure most cases of infantile eczema and that other forms of treatment seem to help very little, supports the external irritant theory.

The type of child with an easily irritated skin is also note



FIG. 34.—Infantile eczema. Note freedom of nasal and orbital areas. (From Jacobi Atlas of Dermochromes London Heinemann.)

worthy in this association. One is struck by the frequency with which eczema is found in infants with fair hair, blue eyes and a fine smooth white skin and who are well nourished and up to or over weight for age.

**Symptoms.** Eczema usually begins in infants before the age of six months, very commonly about the third month. As a rule it begins on the forehead or cheeks as a redness and roughness of the skin. The epidermis, though apparently dry,

will show on close inspection minute fissures from which oozes a clear serum which has dried into tiny ridges. Later the surface becomes hot, red and swollen and covered with minute vesicles. As a result of friction the vesicles rupture and a raw weeping surface is produced. When the serous exudate has dried into crusts the classical picture is presented, i.e. raw areas covered with yellowish crusts with interspersed oozing surfaces and a moist red base. The orbits, nose and mouth are left free and the eruption has a characteristic mask-like distribution. It spreads commonly if aggravated to the scalp, limbs and trunk. From the start itching is a very prominent symptom, being most marked at night. The infant in its effort to allay this rolls its head from side to side on the pillow if the arms have been immobilised.

**Diagnosis.** The mask-like distribution of eczema in early infancy (up to two years) is very characteristic and even when the eruption has spread elsewhere it is most pronounced on the mask area. It is distinguished from impetigo contagiosa by the œdema of the skin, the pin-head vesication and the presence of irritation, all of which are absent in the latter. In impetigo the crusts are large, the distribution irregular and the parts most attacked are about the nose, eyes and mouth. Scabies sometimes mistaken for eczema, only attacks the face when the baby's cheek is directly infected from the mother's breast. A search for burrows and an inquiry into the history of an itching eruption in the mother, nurse or member of the family should decide the matter.

Eruptions about the buttocks of infants are not eczemas.

**Prognosis.** At the age of two to three years there is a tendency for the disease to disappear. With suitable continuous treatment it is possible to cure infantile eczema in a few months, sometimes if seen early and not yet extensive it may be overcome in a few weeks.

Rare cases of sudden death during the course of the disease have been reported.

In a percentage of cases in spite of treatment the disease continues into later childhood and even into adult life.

**Treatment.** *Internal.* The state of digestion should be inquired into, the motions examined and if any error exists it should be corrected.

For extreme restlessness bromides, chloral hydrate or luminal may be given.

*External* This requires to be most painstaking and constant Protection from all irritation is the basis If the crusts are thick and especially if the surface has become secondarily infected with impetigenous crusts these should be removed by a carefully applied starch and borie poultice (see Appendix) To prevent the child from scratching cardboard splints, extending from shoulder to wrist should be applied

A roll of cardboard placed around the infant's arms outside its clothing prevents fraying of the arms and allows the infant freedom of movement but prevents it from bending the elbows and so using its hands on the face and scalp It is a satisfactory and simple form of restraint After removal of the crusts the most generally useful application is zinc oxide paste (zinci ox dr 2 pulv amyli dr 2 vaseline dr 4) This is spread on strips of plain gauze or ordinary bandage and applied carefully like a plaster on the affected parts The thinnest possible layer of cotton wool is applied over this and a mask of butter muslin with holes for eyes nose and mouth placed over this again and kept in position with a bandage applied around forehead and under the chin A fresh dressing is applied twice every twenty four hours At each change the remains of the previous application of paste is gently removed with warm olive oil or liquid paraffin before applying a fresh dressing of the paste

This dressing may be continued for weeks while extreme care is taken that the infant does not have an opportunity of scratching If an exacerbation occurs it is most likely to be due to failure of the treatment in this respect After the acute stage has subsided however it will usually be found an advantage to add tar to the zinc paste and for this purpose White's tar ointment is a good remedy

Crude coal tar	dr $\frac{1}{2}$	} Mix	} Add together
Zinci ox	dr 1		
Pulv Amyli	dr 3	} Mix	
Vaseline	dr 4		

This is applied as was the zinc paste

Fractional doses of x rays are very helpful even in acute cases one sixth to one quarter S B being given at seven to ten day intervals for a total of four to six treatments the longer interval and the lesser number of treatments being employed for the bigger dose

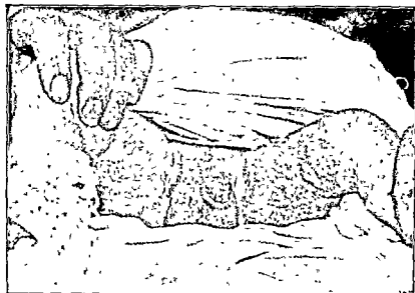


FIG. 35.—Napkin erythema. Actual flexures unaffected. (From Sequeira, "Diseases of the Skin." London: J. & A. Churchill Ltd.)



FIG. 36.—Intertrigo. Eruption confined to flexures and neighbouring skin. (From Sequeira, "Diseases of the Skin." London: J. & A. Churchill Ltd.)

During the entire treatment no soap or water should come in contact with the affected parts

### Napkin Erythema

**Synonyms** Erythema of Jaquet Napkin rash

**Ætiology** Occurs in babies who are suffering from intestinal toxæmia It seldom occurs in infants with normal intestinal flora unless they are cases of neglect It is believed by most to be a form of toxic erythema the site being determined by local irritation of the napkin The *Bacterium Ammonigenes* a saprophytic bacillus derived from the fæces splits urea into ammonia and this is thought to be the immediate cause

**Symptoms** At first there are dark red blotches with a smooth shiny surface confined to the buttocks lower part of the back, perineum, genitals lower abdomen inner sides of the thighs, and very often the back of the calves and heels where these come in contact with the napkin as the baby lies with hips and knees flexed The fact that the flexures which are protected remain free is one of its most striking features At a later stage flat papules one eighth to half inch in diameter appear on the red patches and still later the papules become excoriated, are secondarily infected and ulcerate When ulcerated the condition bears a close resemblance to syphilitic ulceration and it is at this stage that a mistaken diagnosis is made not uncommonly

**Diagnosis** The characteristic feature of the eruption is the occurrence of the rash only on sites which come in contact with an ammoniacal napkin

### Differential Diagnosis

**Napkin Erythema** Occurs only on convexities where napkin comes in contact

**Congenital Syphilis** Occurs on the buttocks very commonly but is associated with other evidences of disease elsewhere

**Impetigo** (see p 316) May complicate the erythema in ulcerative stage but is found also outside the napkin area

**Intertrigo** Affects only the flexures In addition to the groins it may be found in axillæ and folds of neck

**Seborrhæic Dermatitis** May affect the napkin region The

areas are well defined and moist and are covered with greasy scales. Other lesions are found invariably elsewhere.

*Infantile Eczema* Does not affect the buttocks but is found on the face and flexures.

*Treatment Internal* Any errors of diet should be corrected. Half grain doses of grey powder should be given daily for one week and a mixture of sodium bicarbonate three times daily during the same period.

*External* The napkins should be changed frequently and the affected parts washed with warm water without soap. The napkins should be boiled and washed out without soap if rinsed out with 1-4 000 perchloride of mercury and allowed to dry ammonia will not form.

As a dressing a paste of salicylic acid gr 10 pulv zinci or pulv amyli of each dr 2 vaseline to 1 oz will be found generally useful. In the ulcerated type hydrarg ammon 2 per cent substituted for the salicylic acid is valuable. If seen in the early stage of redness a dressing of liquid paraffin will suffice.

### Intertrigo

Intertrigo or chafing is the name given to lesions produced by the friction of two opposed surfaces of the skin.

It is commoner in fat babies but the worst cases occur in neglected infants.

The regions affected are the groins the sides of the scrotum and the flexures of the thighs.

Here the irritation of urine and fæces and improper cleansing of the parts are contributory factors. It is also found in the folds of the neck caused by the irritation of fluid food or dribbling. The friction first produces an erythema and the moisture due to retained perspiration or the fluids already mentioned causes the sodden epidermis to be removed with the result that a raw oozing surface is formed.

Infection by micro-organisms may cause ulceration and spread of the lesions beyond the flexural areas first involved.

*Treatment* This consists in strict cleanliness and avoidance of irritant soaps. After washing a dusting powder made up of equal parts of zinc oxide and talc is useful. If the parts are ulcerated calomel 1 in 10 should be added to the powder. If an ointment is preferred hydrarg ammon gr 5 to 1 or zinci ointment will serve.

## Sudamina (Sweat Rash)

This is common in plump children whose coats or bodies are overlaid or in children who are suffering from pyrexia. The rash is commonest on the chest and neck and is characterised by crops of tiny vesicles rising from normal skin. The vesicles dry up and fall off leaving no stain.

The only treatment necessary is to remove the excess clothing and use a simple dusting powder as recommended for intertrigo.

## ERUPTIONS DUE TO ANIMAL PARASITES

## Scabies

**Ætiology** This is a highly contagious disease common in hospital and dispensary practice. It occurs in epidemics in institutions. It is caused by an animal parasite the *Acarus Scabiei*. The lesions are produced by the female parasite. The parasite burrows into the epidermis to lay her eggs and having done so dies at the distal end of the burrow. The male remains on the surface and is rarely found.

**Symptoms** The chief features of the complaint are marked itching most pronounced at night and the presence of the burrows. These appear as lines one-eighth to a half inch in length grey or greyish black in appearance along which black dots can be observed. At one end of the burrow an opaque spot can be seen barely visible to the naked eye but readily seen with a lens. This is the acarus which can be picked out with a needle and examined with a high power magnifying glass or put on a slide and viewed under the low power of the microscope. Behind the opaque spot a vesicle known as the 'pearly vesicle' is frequently seen. In infants the burrows are not so well formed as in the adult and sometimes a prolonged search has to be made before a typical one is found. They are found between the fingers the ulnar sides of the wrists the sides of the feet under the malleoli and between the toes. In infants the feet are much more frequently affected than in the adult and are often involved when the hands are not. In the adult the face is never affected and in the infant rarely.

In addition to the burrows there are usually scratch marks scattered papules over the trunk and limbs with secondary 'eczema' and impetigo and/or pustules.

**Diagnosis** A child with a general itching eruption has either scabies or papular urticaria (lichen urticatus the gum)

The diagnosis between the two is sometimes very difficult. The finding of a typical burrow with the parasite removes all



FIG. 37.—Scabies in an infant. Bullæ on feet which are markedly affected. (From Sequeira *Diseases of the Skin* London J & A Churchill Ltd.)

doubt. If this is not possible a history of itching eruptions in other members of the family is strongly suggestive of scabies.

An examination of one of these persons will often disclose a typical burrow. Wheals with central papules situated on the buttocks suggest papular urticaria. All patients with generalised "eczema" and impetigo about the hands and

feet should be suspected of scabies and a thorough search made for a burrow

**Treatment** The object of the treatment is to destroy the acarus not only on the skin but also in the clothing and bed clothing of the baby. Sulphur ointment (B P), which is so useful in the adult, is too strong for the tender skin of the infant and tends to set up a sulphur dermatitis. However, it may be employed in strength gr 10 to the ounce. Mitigal (Bayer) an organic preparation of sulphur does not produce a dermatitis, and is a reliable remedy.

The treatment must be very thorough and carried out with meticulous attention to detail. The infant is given a bath of warm water and soft soap. It is then rubbed over from chin to toes with mitigal special attention being paid to the folds. This is repeated for three successive days when if the treatment has been carried out thoroughly the baby will be cured. If any eczematous or impetigenous lesions remain treatment can be continued with an ointment of 2 per cent B naphthol in vaseline. The secondary eruptions clear up rapidly if the scabies is cured.

**Disinfection of Clothing** All bed clothes and underclothing must be thoroughly disinfected. Cotton or linen articles are best disinfected by boiling. Woollens and flannels may be disinfected by soaking for twenty four hours in strong solution of evlin and afterwards washed in cold water. Outer garments may be disinfected by dry heat and this will be done in any large town by the Public Health Department. If no such facilities are available the clothes can be hung in the open air for a couple of days after spraying with formalin.

### Pediculosis

Pediculosis corporis is very rarely seen in children. Pediculosis capitis commonly seen in older children is not common in infants. If nits and head lice are present frequent washing with soap and warm water will suffice in most cases. If there is accompanying impetigo the hair should be cut short and, after washing with soap and warm water, an ointment of ungt hyd nit dil (B P) vaseline aa rubbed in twice daily for a few days. If the case is severe, the scalp should be mopped over with oil of sassafras and afterwards washed with soap and warm water to which has been added a little paraffin oil.

## Fleas

The appearance of bites of the common flea (*Pulex irritans*) is so well known as to require no description. The dark puncture with the surrounding hæmorrhagic area must not be mistaken however for the eruption of purpura simplex.

## ERUPTIONS INFECTIVE IN ORIGIN

## Streptococcal Infections

Under this heading may be placed —

- (1) Bullous impetigo wrongly called pemphigus neonatorum
- (2) Dermatitis exfoliativa neonatorum (Ritter's disease)
- (3) Common impetigo contagiosa of Tilbury Fox
- (4) Impetigo—intertrigo in type
- (5) Chronic impetigo or impetigo pityrodes
- (6) Ecthyma
- (7) Erysipelas

## Bullous Impetigo of Infants

**Ætiology** It begins as a rule on or near the umbilical stump before it has healed though a small number of cases appear to start in the napkin area due to infection through a microscopic opening. The infection is not uncommonly transmitted by the nurse or mother either of whom may be suffering from an ordinary impetigo or still more commonly from a whitlow. In some cases other children in the family have common impetigo.

**Symptoms** The bullæ or blisters usually start close to the umbilical stump. They spread rapidly over the abdomen, buttocks, thighs and trunk. The limbs usually escape and so does the face and scalp. The horny layer being thin the bullæ rupture early and there is left a bright red oozing surface. In a good number of cases the infant becomes very sick, turns a jaundiced colour and often dies. In such cases the infection has probably entered the umbilical vessels and passed backward to the liver setting up acute hepatitis associated with jaundice. The writer has seen such a case at post mortem with pus in the umbilical vessels and urachus.

The diagnosis from congenital syphilis of the bullous type rests on the bullæ in this case being bigger and usually starting on the abdomen with the limbs escaping. In syphilis the

palms, soles and buttocks are affected. In addition there are coppery macules and perhaps other syphilitic manifestations which are not present in bullous impetigo.

Prognosis is always grave. The mortality in cases requiring admission to hospital (i.e., extensive cases) being as high as 30 per cent.

#### **Dermatitis Exfoliativa Neonatorum (Ritter's disease)**

The rare disease described by Ritter von Rittersham, of Prague, is believed to be a modification of bullous impetigo neonatorum. Instead of blisters rising it creeps under the skin, which is shed in flakes. It begins in the first five weeks of life, usually in institutional children. It starts as an erysipelas like eruption round the mouth and spreads rapidly all over the body. Usually some bullæ rupture and crust.

The mortality is very high, being brought about by complications mostly of the chest and intestine. It is a very rare disease in this country. Some of the very extensive cases of pemphigus neonatorum where large areas of the body are denuded, are sometimes referred to as Ritter's disease, but this is incorrect. Such cases in no way correspond to the disease, as described by Ritter.

**Treatment** The most important treatment of bullous impetigo of infants is prophylaxis. Midwives with septic lesions or whitlows should not be allowed to practice. When a case occurs the mother and midwife should be carefully examined, also any member of the family coming in contact with the infant. It is not uncommon to find several cases in the practice of a midwife who is unaware of the presence of a superficial whitlow or a septic onychia.

When the bullæ are present the infant should be put in a warm bath of 1 per cent. boric acid. When in the bath all unruptured blebs are opened, tags of loose skin snipped off, leaving no pockets. A sterile towel is used to dry the infant, who is then swathed in sterile lint spread over with boric ointment or zinc cream to which has been added 1 per cent. gentian violet or malachite green. Another good method is to swab over the surface with 4 per cent. silver nitrate in spirit and then powder. If the bullæ are small and fresh they may be injected with 4 per cent. silver nitrate as soon as discovered.

## Common Impetigo Contagiosa of Tilbury Fox

**Ætiology** Very common in hospital and dispensary practice. Several children in the same family or school may be affected.

**Symptoms** The eruption usually occurs on the face, beginning around the nose and mouth. At first the lesions are clear



FIG. 38.—Impetigo Contagiosa. Acute stage. (From Carder's Handbook of Skin Diseases. Lilliburg, F. & S. Livingston.)

vesicles or blisters. In a few hours they become muddy, rupture, dry, and form large amber-coloured crusts. The crusts have a "stuck-on" appearance and can be readily removed.

with forceps. In most cases it will be possible to see all stages of the eruption : *e* clear vesicles muddy vesicles and the typical crusts. The eruption is extremely auto inoculable and may spread rapidly over the face and scalp. The disease usually lasts about three weeks from its first appearance when fresh lesions cease to form and the crusts dry and fall off.

The glands draining the affected area may become inflamed and even suppurate.

**Treatment** The first step consists in removing the crusts. This may be done in mild cases by bathing with solution of bicarbonate of soda. In more widespread cases by the application of frequent boric fomentations or a starch and boric poultice. When the crusts are removed mild antiseptic ointments are applied. The ointment should be made thick so that it will remain on. For this purpose ungt. hydrarg. ammon. to which has been added zinc oxide powder dr. 2 to the ounce will answer. On the scalp where it is important to prevent spread early lesions can be successfully aborted by the daily application of silver nitrate gr. 20 to the ounce of industrial spirit.

### Intertrigo Type of Impetigo

**Intertrigo Type** This is an impetigo found where intertrigo occurs : *e* in the flexures. It is however also seen in the post auricular sulcus where intertrigo does not occur. This type of impetigo is frequently referred to as post auricular eczema. When ordinary impetigo occurs the lesions are at first clear vesicles or phlyctenules. In a couple of hours the contents become muddy with pus dry up and form the characteristic yellowish crust which appears to be stuck on and which can readily be removed with a forceps. In the intertrigo type occurring in the flexures the horny layer being thin the phlyctenules rupture immediately leaving a moist eczematous surface. The contagious contents of the phlyctenules flow over the neighbouring skin and produce typical crusted impetiginous lesions in the immediate neighbourhood. It differs from an eczema and from a dermatitis in not being itchy having no vesicles and there being no swelling of the skin.

The treatment consists in removing the crusts drying the moist parts with a lotion of equal parts of hydrogen peroxide and calamine lotion, and when dry treating as a common

**impetigo** Fissures are very liable to form and these should be painted with 2 per cent silver nitrate solution

### Chronic Impetigo

In children of low resistance when the crusts of ordinary impetigo have disappeared there remain circular scaly patches. These are frequent on the face especially on the chin at the angles of the mouth which are frequently fissured. Several names are given to these conditions impetigo pityrodes

perleche etc. A similar condition is found on the upper lip of children suffering from nasal discharge and in front of the ear in children with chronic otorrhœa. There is no doubt that they are all of coccal origin and require comparatively strong remedies for their removal one of the best being oil of cade ℥ 30 resorcin gr 15 ungt hyd ammon ad oz 1 well rubbed in twice daily. Cod liver oil generalised sunlight good food and change to the country help in clearing up the disease

### Ecthyma

In poorly nourished and debilitated children impetigo lesions may take a more serious form. Ordinarily impetigo is an extremely superficial disease occurring just beneath the horny layer of the skin which on healing leaves no scar. In ecthyma however the impetigenous crusts are a dirty brown colour with a crop of vesicles around them. When the crust is removed there is a shallow punched-out ulcer with a red halo round it. This type of impetigo is generally one which occurs secondarily in some itching eruptions such as scabies pediculosis or papular urticaria. The common sites are the buttocks and lower abdomen. When healed chronic infiltrated patches remain resembling a tuberculous lesion. The condition is fairly easily recognised—a debilitated child—perhaps some itching eruption present—most likely typical impetigenous lesions which have not ulcerated—multiple small lesions on the abdomen or buttocks.

The ulcers are difficult to heal and treatment must be thorough. If there is an accompanying itching eruption it must be treated *secundum artem*. The crusts are removed in the ordinary way. Ulcers washed with 1:3000 perchloric. After disinfection the ulcers should be painted with silver nitrate gr 15 in an ounce of spirits ætheris nit.

Most important of all the diet must be corrected and the general health of the baby attended to. Cod liver oil and generalised ultra-violet light baths are useful both for their tonic and general bactericidal effects.

### Staphylococcal Infections

#### *Impetigo of Bockhart*

This is an infection of the pilo sebaceous follicles by the *staphylococcus pyogenes aureus*. The term includes all staphylococcal infections of the follicles from small pin head sized pustules to large boils. In infants it is usually met with —

(1) Associated with itching eruptions such as scabies or papular urticaria

(2) Secondary to impetigo contagiosa

(3) Following the application of poultices or other dirty dressings on abscesses or boils



FIG. 39.—Bockhart's impetigo of thigh (Staphylococcal). The lesions are small abscesses centred by a hair and surrounded by a zone of erythema. (From Sequeira 'Diseases of the Skin' London J. & A. Churchill Ltd.)

**Treatment.** Frequent bathing with hot water and painting with brilliant green 1 per cent in 25 per cent spirit is usually sufficient to cure the milder cases. The larger lesions should be fomented with boric lint and incised. General ultra-violet baths to raise the patient's resistance and the administration of a staphylococcal vaccine in stubborn cases is recommended.

### Multiple Subcutaneous Abscesses in Infants

This type of staphylococcal infection occurs in infants who may be suffering from common impetigo contagiosa Bockhart's impetigo eczema or other pruritic eruption. In some cases, however the child appears otherwise normal.

The lesions are numerous intradermic or hypodermic nodules the size of a pea or larger. The skin over many of them is red while in others it appears normal.

The swellings are elastic and when incised a thick creamy pus is evacuated. The baby's general condition is variable some of the infants being in a fairly healthy state without pyrexia while others are gravely ill with all the evidence of a septicæmia.

The peculiarly widespread distribution of the lesions and the presence of normal skin over many of them points to a blood stream infection. This is difficult to reconcile with the cases who appear to be in fairly good health.

**Treatment.** Any accompanying impetigo or other skin condition should be treated in the ordinary way. Antiseptic boric baths should be given. When in the bath incision of the abscesses is recommended.

General ultra violet light baths are very valuable and the internal administration of quinine apparently helpful. In the absence of septicæmia the prognosis is good.

### Seborrhœic Dermatitis or Eczema Seborrhœicum of Infants

The term seborrhœa is applied to a number of diseases varying from the one under consideration to the dry scaly dandruff of later childhood and adults. It appears to be established that the pityrosporon of Malassez (bottle bacillus of Unna) given favourable conditions on the part of the host is responsible for the production of the lesions described. One of the favourable conditions is an oily or greasy medium.

Children affected by seborrhœic dermatitis have the remains of the vernix caseosa over the anterior fontanelle which mothers are often afraid to wash fearing injury to the brain. This caseosa is referred to as cradle cap. It will be found on inquiry that most of these children suffering from seborrhœic dermatitis have or have had a cradle cap. In seborrhœic dermatitis irregular patches or one large patch of yellowish

greasy looking scales are found on the vertex. On the face especially in the naso labial folds extending on to the cheek on the neck and behind the ears there are sharply defined reddish areas covered with yellowish scales in parts raw and oozing.

In the groins are similar areas or the whole naphin region may be covered with one large red raw or scaly area. The eruption comes out quickly and spreads rapidly. Often the mother or nurse is also found to be affected with seborrhoea of the scalp.

The diagnosis has to be made from infantile eczema and naphin erythema.

For differential diagnosis see p. 308.

**Treatment.** Once daily the parts should be sponged over with warm water and a superfatted soap. All traces of soap must be removed before drying. An ointment of salicylic acid gr 2½-5 sulph precipitat gr 5-10 to the ounce of vaseline should be rubbed into the scalp twice daily and an ointment of sulph precipitat gr 5-10 to the ounce of zinc ointment will clear up the eruption rapidly. This rapid clearance under mild sulphur treatment is one of the features of the condition. After clearing strict cleanliness of the scalp prevents recurrence.

### Molluscum Contagiosum

This is caused by a filterable virus. It is contagious and the eruptions may occur in great numbers on the same patient from auto inoculation. The lesions are hemispherical papules or flat button like discs of a milky white or pink colour. There is a depression in the centre of each giving it an umbilicated appearance. On compression between the thumb nails a semi solid white mass exudes from the central orifice. The tumours vary in size from a pin's head to a large pea.

The face eyelids neck and genital organs are the parts most often affected.

If untreated they last indefinitely but give rise to no symptoms. Sometimes they suppurate from secondary infection with pyogenic organisms.

**Treatment.** Each wart should be incised and the contents squeezed out. Hæmorrhage ensues this is easily checked by pressure. The inside of the sac may be touched with a pointed match-stick dipped in 1-20 carbolic acid solution.

### Thrush (*Monilia Albicans*)

The thrush fungus occasionally traverses the alimentary canal and gives rise to an eruption in the naphkin area. The lesions are reddish macules oval in shape peeling at the edges with the free edge of the peeling directed towards the centre. The presence of thrush in the mouth or a history of recent infection will confirm the diagnosis. Application of an ointment of salicylic acid and benzoic acid (3 per cent of each) will clear up the condition in about a week. An alternative treatment is gentian violet 2 to 3 per cent in 25 per cent spirit.

### The Skin Eruptions of Congenital Syphilis

Congenital syphilis has been dealt with elsewhere and will only be considered here with reference to its cutaneous manifestations. These are best considered under two heads —

(1) Hereditary syphilis with cutaneous lesions visible at birth

(2) Hereditary syphilis the child being born apparently healthy but showing signs of syphilis at a later date

(1) *Hereditary Syphilis with Cutaneous Lesions Visible at Birth* The skin lesions consist of flaccid bullæ on the palms and soles set on a reddish coppery base which is bigger than the bullæ. The blisters contain serum or sero pus and are symmetrically distributed. This is the so-called pemphigus syphiliticus infantum. The bullæ are usually confined to the palms and soles rupture and expose a red moist base or exude the sero pus which may dry and form crusts. As a rule these children die within a few days—often suddenly. If they survive they may develop other cutaneous syphilitic lesions such as fissured mouth condylomata around the anus and umbilicus etc. The diagnosis of such a case presents little difficulty. The bulla are differently distributed from those of bullous impetigo neonatorum in the latter the palms and soles escape. Pustular scabies may provide a somewhat similar picture. The age of the infant the presence of scabies on its attendants the finding of burrows will distinguish between them. If still in doubt the presence of spirochetes in the bullæ and a positive Wassermann will decide the diagnosis.

(2) *The Child being Born Apparently Healthy but showing*

*Signs of Syphilis at a Later Date* This is by far the most important group. The cutaneous manifestations appear from the second week to the end of the third month. As a rule they are papular bullæ being rare in this type. The eruption

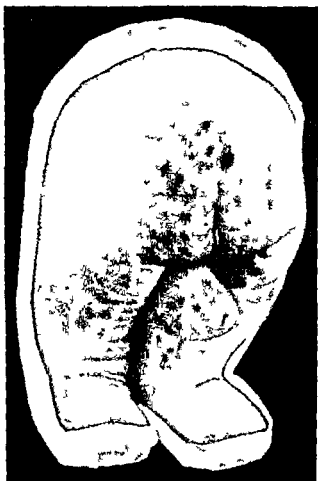


FIG. 1.—Congenital syphilis. A common site. (From Jacoby, *Atlas of Dermatology*. London: Reimann.)

occurs on the buttocks, thighs, legs and feet, and sometimes on other parts of the body. The papules are round, rose pink or dusky red in colour. In moist warm areas such as the genital crural they may hypertrophy and assume the character of condylomata. Some of the papules, especially on the convexities of the buttocks, may ulcerate, and when they disappear they leave brownish stains. The lips usually fissure,

and a little condyloma may be found at each angle of the mouth. The nails may be affected and ungual and peri ungual inflammation may lead to their loss. The eruption is therefore more or less polymorphic and closely resembles that seen in the secondary stage of acquired syphilis.

The diagnosis of the condition should not be difficult. No other condition at this age presents a similar polymorphic eruption. There will probably be co-existent evidence of syphilis in other organs.

The diagnosis from Jacquet's napkin erythema and scabies will be found under these headings.

### Herpes

*Herpes roster* is very rarely seen before adolescence. *Herpes febrilis* (*herpes recurrens*) is an eruption of vesicles seen about the lips and nose usually in association with the common cold. It occurs with various other febrile infections particularly tonsillitis pneumonia cerebrospinal meningitis and with gastric disturbances. It may also occur upon the cheek chin neck ear and buttock. It tends to recur at intervals and usually affects the same area at each recurrence.

The eruption consists of tiny vesicles the size of a pin's head on an erythematous base. After a few days the vesicles dry forming a scab which later falls off leaving no or at most only a slight scar.

**Ætiology** It has now been definitely proven that this disease is caused by a filterable virus which lives normally a semi-saprophytic existence on the mucous surfaces and that a particular strain of the virus is the causative agent of encephalitis lethargica. Peripheral irritation as from an erupting tooth appears in some cases to determine an attack.

**Treatment** In the recurring type a search for possible cause should be made.

The essential point in the treatment is to cover the lesions and protect them from irritation. This may be done by a powder of zinc oxide and starch or talc.

Some measure of success has been achieved in recurrent cases by inoculation. The arm is scarified as for vaccination and the contents of the fresh vesicles inoculated. The vesicular lesion produced is in turn inoculated on a fresh scarification mark and the process continued as long as it is possible to secure a success.

(1) Local irritation, e.g. the sting of a nettle bites of insects jelly fish etc

(2) Toxic bodies from without

(i) Some foods are toxic to certain individuals  
e.g. oatmeal strawberries tinned fish,  
eggs etc

(ii) Drugs quinine salicylates mercury and  
several others

(iii) Intestinal parasites

(3) Toxic bodies developed in the body

In children these are usually formed in gastro intestinal disorders

The onset of urticaria is acute sometimes with a slight degree of fever but usually without

There may be evidence of gastro intestinal irritation—vomiting and diarrhoea

The eruption consists of well-defined white or pink swellings. The margin is often red with the centre pale. It exactly resembles the wheal produced by the sting of a nettle. The itching is intense and the scratching it induces brings out fresh wheals. A special characteristic of the urticarial wheal is its rapid development and its equally rapid disappearance leaving behind no scale or stain.

### Urticaria Papulata

Synonyms Strophulus gum rash (in Dublin the gum) urticaria chronica infantum lichen urticatus prurigo simplex

Ætiology *Urticaria papulata* is peculiar to infancy and childhood. It is so common that few children escape it in some degree. It occurs between the ages of six months and two years, i.e. roughly during the period of the first dentition.

It occurs most commonly during the later months of spring and early months of summer.

Hallam has shown that it is an allergic manifestation and believes that the home sleeping arrangements have something to do with it. This however is doubtful for a series of experiments have been carried out by admitting children to hospital having brought with them their own beds bed-clothing etc and having their usual food supplied from their homes. In addition some of the plaster from the walls and the dust from their rooms was scattered around their beds. In spite of

thus the children got rapidly well in hospital without treatment of any sort

**Signs and Symptoms** The rash appears most commonly on the extensor surfaces of the limbs. The buttocks, sacral region

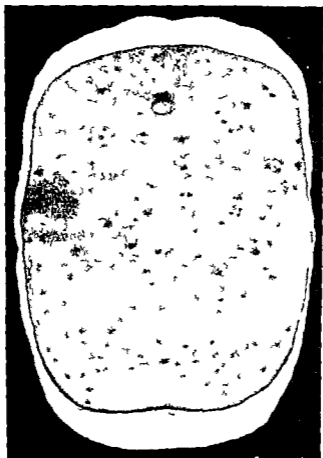


FIG. 41.—Papular urticaria. Multiple papules many of which are tipped with hemorrhagic crusts. (From Jacobi's Atlas of Derm ochromes. London: Heinemann.)

and lower abdomen are frequently involved. The face, backs of the hands and feet, chest and back usually escape. Under careful observation a primary wheal of the ordinary urticarial type is seen. This fades in an hour or so but leaves behind a papule. The papule at first red, later becomes the colour of the surrounding skin. Because of scratching the papule frequently carries a hæmorrhagic tip, in old standing cases papule and surrounding skin are pigmented, presenting a light or dark

brown colour. The itching is most intense at night and the mother insists that the child never sleeps. This information is not consistent with the appearance of the child who is usually plump and well nourished.

Secondary manifestations sometimes appear in the form of impetigo or septic dermatitis in which case the lymphatic glands become enlarged.

The distribution of the eruption is usually symmetrical. Fresh crops appear at intervals usually coming up at night.

**Prognosis.** The condition is difficult to cure but the children usually improve in cold weather and lose the eruption completely as they grow older. A number of cases pass into the more serious condition of prurigo. The disease is believed by many to be a connecting link between urticaria and prurigo.

**Diagnosis.** The diagnosis must be made from scabies, varicella and prurigo of Hebra (see Table VII).

**Treatment of Urticaria.** In the acute variety careful inquiry must be made with the object of discovering any offending article of food such as strawberries, eggs, fish, etc. Also an inquiry regarding the administration of drugs, vaccines and serums.

A dose of calomel or grey powder followed a few hours later with milk of magnesia or other saline is often useful. A bath to which sodium bicarb. one teaspoonful to the gallon has been added helps to allay itching.

The skin should be patted dry with a soft sheet and the infant clothed in silk or cotton (not woollens or flannels). As external application various anti-pruritic remedies may be used and it is often necessary to vary them or alternate them. Amongst some of the useful ones are calamine lotion to which has been added 2 per cent phenol. B. naphthol gr. 10 to ounce of vaseline or 1 dr. to 10 oz. of water of any of the following sodium biborate, sodium carbonate, liq. plumbi subacet, liq. picis carbonis.

In severe cases the baby is best kept entirely in its cot avoiding dressing or undressing as much as possible.

In the *papular form* any defect in the general health must be corrected. Dyspepsia and constipation specially require attention. The presence of intestinal parasites and external parasites such as the common flea should be sought for. Clothing and bathing as recommended in the acute form are helpful. A mixture of rhubarb and soda with small doses of calomel or

Table VII

Disease	Age of patient	Nature of eruption	Site of eruption	Infectivity	Prognosis
<i>Acute urticaria</i>	1 to 2 years	Widespread papules	Extremities of limbs and buttocks	Non-contagious	Marked
<i>Sealed urticaria</i>	Any age	Burrows & vesicles	Limbs and feet & face free	Contagious	Marked
<i>Varicella</i>	Any age	Vesicles	Trunk, face, scalp and limbs in usual order of appearance & sometimes mouth	Infectious	Variable, but usually slight
<i>Herpes zoster</i>	Sometimes in first year but not marked till age of 3 years	Papules on yellow skin later vesicles	Extremities buttocks trunk and face	Non-contagious	Violent

grev powder as additional purgatives and intestinal antiseptics appear to be useful

If the child sleeps on feathers the substitution of these by kapok sometimes meets with success

The external applications as outlined for the acute form will suffice B naphthol being the most generally useful in this form especially if the lesions are septic Should all these remedies fail transference of the child to hospital or to a new environment will almost always succeed in effecting a cure

### Drug Eruptions

*Bromide of potassium* is frequently given to infants on medical advice or as patent soothing powders

A fair percentage of children are susceptible to the drug and an eruption may be produced by very small doses The eruption begins as a vesicular eruption but the vesicles soon become firm and opaque project above the surface in tense button like lesions from the size of a split lentil to an inch or more in diameter It usually occurs on the face shoulders and legs and when fully developed presents a striking picture After a few days the lesions dry and form crusts which fall off leaving no scars If the drug is continued large fungating granulations form

In the early stages the rash might be confused with varicella but when fully developed can hardly be mistaken The treatment consists in withdrawing the drug

*Antipyrin* produces a rash closely simulating measles but the other symptoms of measles are absent

*Belladonna* either taken internally or applied externally may produce a rash similar to scarlatina The rash is usually confined to the shoulders and upper part of the trunk Other symptoms of belladonna absorption such as dilated pupils and dry mouth are present The rash is frequently followed by desquamation

Other drugs frequently given to children and which produce rashes are *chloral* (scarlatiniform with desquamation) *luminal* (itching erythema and wheals) *quinine* (scarlatiniform with desquamation and sometimes pyrexia—rash itches) *pyra milon* (erythema sometimes purpura) *santonin* (urticarial) *turpentine* (urticarial) The treatment is to withdraw the offending drug and allay irritation if any exists

### Enema Rash

This is not common in children below the age of six years. It is probably a toxic rash derived from some substance in the feces which is absorbed when a large quantity of water is injected. It is not as was once thought due to soap for it follows with equal frequency plain water enemata. It usually occurs twelve to twenty four hours after the administration of the enema but may come on in two to three hours or be delayed for thirty-six to forty two hours. The rash is scarlatiniform, morbilliform or urticarial and involves chiefly the thighs, buttocks and lower abdomen.

### Serum Eruptions

These eruptions are most common after subcutaneous injections of anti diphtheritic, anti streptococcal or normal horse sera. The eruption may occur in the course of a few hours or be delayed for one or more weeks. The phenomenon is anaphylactic and the worst outbreaks are in those already sensitised by a previous injection ten or more days previously. The common form of eruption is large urticarial blotches with a somewhat generalised distribution. It may however be morbilliform or scarlatiniform. There is usually a rise of temperature to  $101^{\circ}$  to  $102^{\circ}$  F. and frequently joint pains.

The treatment consists in the injection of adrenalin and/or the administration of calcium and parathyroid substance.

### Vaccinia and Vaccination Eruptions

Ignorance and prejudice attribute to vaccination a large number of cutaneous affections in infancy.

It is therefore important to be familiar with the conditions which may be caused by vaccination and those which may reasonably be ascribed to it. It should be clearly understood that calf lymph is obtained from animals proved to be free from tuberculosis and the lymph is sent out only after a careful post mortem has been carried out on the calf. It is impossible for the tubercle bacillus to live in glycerinated lymph and it is impossible for the calf to convey syphilis.

### Eruptions Caused by Pure Vaccine

The normal process following inoculation with calf lymph is

too well known to warrant description. It sometimes happens however that as a result of scratching or other injury redness and swelling are not limited to the area around the vesicles but spread until the greater portion of the arm and shoulder is affected. If cellulitis occurs mixed infection will be found.

The treatment consists in putting the arm at rest using soothing lotions of calamine or lead if the eruption is erythematous and borie fomentations if there is ulceration or secondary infection present.

### Auto-inoculation

This takes place from scratching the primary vesicles before they have healed and hence may occur as late as the tenth day after vaccination. In most cases the sites of reinoculation have been areas of impetigo herpes varicella etc. It is hence inadvisable to vaccinate a child who is suffering from any skin affection.

### Generalised Vaccinia

This name is wrongly applied to extensive vaccinia caused by auto inoculation as just described. There are however cases where a true generalised vaccinia does occur. From four to nine days after vaccination a crop of lesions come out which pass through all the stages of normal vaccination i.e. papule vesicle and in these cases pustules. The affection lasts for about three weeks may be afebrile or febrile depending on the extent of the eruption.

No special treatment is required.

### Toxic Vaccination Rashes

These rashes do not differ from the other toxic eruptions already described. Eruptive erythematæ are not uncommon during the evolution of the vesicle i.e. from the fourth to the tenth day. They may be roseolar morbilliform scarlatiniform urticarial or resemble an erythema multiforme. Occasionally a bullous eruption occurs but usually after the vaccination has healed. All are of rare occurrence and require no special treatment.

### Eruptions of Doubtful Connection

There is no evidence that any of the common skin diseases

of infants such as eczema papular urticaria etc are ever caused by vaccination Psoriasis has been known to start in



FIG. 4. —Vaccinal bullous erythema. (From Sequerra Diseases of the Skin London J & A Churchill Ltd.)

vaccination scars In predisposed persons we know that it also starts in wounds and slight injuries and hence the vaccination wound is no exception It is a very rare complication

Sclero œdema and sclerema are dealt with on p 61

### Care of the Normal Skin

Normal or approximately normal skin of an infant requires no more care than is likely to be given to it by the nurse or the mother. Generally speaking normal skin suffers from too much attention.

The new born baby may be cleansed for a couple of days with olive oil and for a longer period if premature or unhealthy. Unless there is some contra indication it can afterwards have a daily sponge bath with warm water and a neutral soap. The soap should be at all times completely removed by thorough rinsing before the infant is dried.

After drying it is a good plan to rub in a little olive oil. The scalp stands soap and water very well and this should be used daily until all the vernix has disappeared. Afterwards it is sufficient to wash the scalp twice weekly while a daily bath is given to the rest of the body. Frequent washing of the buttocks is obviously necessary but these parts stand soap and water well especially if after washing they are rubbed over with olive oil or dusted with talc. If the parts become irritated they are best cleansed with olive oil.

Oily or greasy skins stand soap and water well and after bathing should be dusted over with talc.

Dry skins do not tolerate soap and water so well and after washing should be rubbed over with olive oil.

If the skin becomes irritable and chapped it may be necessary to cleanse temporarily with olive oil and the parts such as the face feet and hands may be cleansed with the following lotion

R Magnesiæ pond	oz 4
Paraffini liq	oz 2
Sodii boratis	dr 1
Aq rose	ad oz 8

It is generally believed that the skin of the average child gets sufficient light and air during the daily routine. In cold weather it is advisable however to insure that a large portion of the child's body is exposed to air and daylight (sunlight not necessary) for half an hour daily in a warm well ventilated room.

## APPENDIX

## Baths

*Boric Bath* Made by adding 2 oz. of boric powder to each gallon of water. Soothing, antiseptic and mildly astringent.

*Starch Bath* Made by adding  $\frac{1}{2}$  oz. of starch to each gallon of water. Soothing and antiphlogistic.

*Sodium Bicarbonate* (Common bread soda) Made by adding 1 drachm to each gallon of water.

Anti pruritic and solvent of scales and crusts.

## Poultices

*Starch and Boric Poultice*

One drachm of boric powder }  
One oz. of starch } Mix to a cream with cold water

Add water, which must be boiling to 1 pint. When cool spread in layers  $\frac{1}{4}$ – $\frac{1}{2}$  inch thick between folds of gauze. Apply on affected parts and cover with oiled silk or waterproof sheeting and leave on for twelve hours.

When well made and well applied is the best method of removing crusts from inflamed areas of eczema, dermatitis or the crusts of *impetigo contagiosa*.

Is soothing, antiseptic, mildly astringent and antiphlogistic.

*Linseed poultices* should not be applied in skin eruptions. They are dirty and when applied to boils and abscesses promote spread of infection, giving rise to the *impetigo* of Bockhart.

*Boric Foment* Made by cutting strips of boric lint larger than the area to be treated. The strips of lint are rolled in a sterile towel which is put into a large bowl. The ends of the towel are allowed to hang over the sides of the bowl. Boiling water is poured into the bowl and after a few minutes the dry ends of the towel are caught and the boric strips within wrung dry. The lint is turned out rapidly and applied while hot. It is now covered with oiled silk or other material impervious to water. The material placed over the lint should be bigger in area than the strip of lint. The whole is covered with a layer of cotton wool and fastened with a bandage.

It makes an excellent dressing for the purposes indicated under 'starch and boric poultice,' but is in addition a good and suitable dressing for boils, abscesses and localised infections generally.

The foment should be renewed every couple of hours, always using fresh lint.

Other fomentations can be used by wringing sterile gauze out of the particular solution, such as 1–3,000 biniodide or perchloride of mercury (valuable where there is secondary infection).

*Exsol* (also for infected areas), etc.

In these cases no oiled silk or such like covering should be used.

## CALAMINE LOTION

R Calaminæ præparata	gr 30
Zinci ox	gr 15
Glycerini	℥ 30
Aqua calcis	ad ℥ 1

Soothing, antiphlogistic lotion suitable for use on acute erythematous eruptions

To it may be added —

- (1) Phenol gr 5 for relief of itch in urticaria toxic erythema etc
- (2) Sulphur præcipitat gr 10 for use on moist areas of seborrhœic dermatitis
- (3) Ichthyol gr 10, for erythematous eruption with swelling of the skin
- (4) Boric powder gr 10 for areas mildly infected and for its anti pruritic and astringent properties

*Zinc Cream*

R Zinci oxide	℥ 3
Adeps lanæ hydros	℥ 1
Ol amygdalæ } Aqua calcis }	aa ad ℥ 1

Soothing Cream Suitable dressing for irritated buttocks

*Zinc Paste (Lassar's)*

R Pulv zinci oxide } Pulv amyli }	aa ℥ 4
Paraff moll ad	℥ 1

Soothing and drying paste Suitable in infantile eczema and napkin erythema Very useful as a base to which can be added, according to indications —

Salicylic ac	gr 10
Sulphuris præcipitat	gr 10
Hydrarg ammon	gr 5-15
Liq carb deterg	℥ 10-30

*White & Coal Tar Ointment*

R Crude coal tar	℥ 1	} Mix
Zinci ox	℥ 2	
Pulv amyli	℥ 1	} Mix
Paraff moll	℥ 4	

Add together

A useful dressing in stubborn cases of infantile eczema

*Beta Naphthol Ointment*

R B naphthol	gr 10-20
Paraff moll	ad ℥ 1

Useful ointment in papular urticaria and to clear up the septic lesions of scabies

## SECTION VII

### CHAPTER XXVIII

T. GILL

#### GENERAL SURGICAL DISORDERS

(*Acute Intussusception—Hirschsprung's Disease—Spina Bifida—Acute Osteomyelitis—Acute Arthritis of Infants—Developmental Defects of Rectum and Anus—Phimosis—Paraphimosis*)

##### *Acute Intussusception*

An intussusception is the prolapse of one piece of intestine into the adjoining part as a rule the condition is steadily progressive

**Morbid Anatomy** A simple intussusception consists essentially of three tubes embracing one another the outer or ensheathing layer, the middle or returning layer and the inner or entering layer. The ensheathing layer forms the *intussusciens* and joins the returning layer at the neck of the intussusception. The entering and returning layers constitute the *intussusceptum* and meet at the apex or distal part of the invagination. In vertical section the mass consists of six layers, three on each side of the central canal, and on transverse section of three concentric rings, so arranged that mucous surfaces are in contact with mucous surfaces and serous with serous.

As the intussusception increases in length the mesentery is dragged in between the entering and returning tubes which thus become curved with the concavity towards the mesentery. The dragging in of the mesentery causes constriction of the veins, and this leads to extreme congestion of the intussusception which becomes purple in colour. As the congestion increases, blood is extravasated into the coats of the bowel and an excess of mucus, mixed with blood oozes from the mucous surfaces and is passed by the rectum.

The effects of the congestion are most marked in the returning tube, and towards the apex of the intussusception which may become very swollen and thus, together with the adhesions

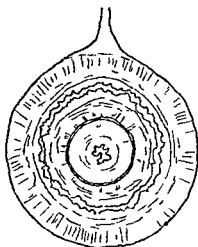


FIG. 43.—Cross-section of Intussusception. Showing three concentric rings and arranged with mucous surfaces in contact with mucous surfaces and serous with serous.

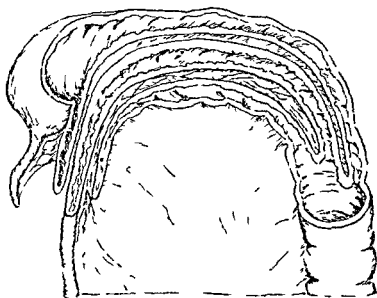


FIG. 44.—Vertical section of Intussusception. (Modified from R. C. Coffey.) Showing sheath returning tube and entering tube. The apex is formed where the entering and returning layers join. The mesentery is drawn into the neck on the concave side of the intussusception.

formed between the opposed serous surfaces, eventually renders the intussusception irreducible. Finally, the œdematous and congested intussusception is attacked by bacteria, and gangrene may supervene. Ulceration and gangrene of the outer layer is rare.

**Mechanism of Production.** The modern view is that swelling of the pre-existing lymphoid tissue in the lower ileum acts as a foreign body and produces spasmodic contraction of the bowel around it, with inhibition of that part immediately distal. The conditions are therefore suitable for the contracted part to pass into the dilated portion beyond. Similarly, a polypus or inverted Meckel's diverticulum may cause irregular peristalsis with spasmodic contraction of the bowel around them, yet they themselves will not form the apex of the intussusception.

**Varieties.** There are three main types in infants. (a) *Ileo-cæcal*, the ileum passes into the colon with the ileo-cæcal valve as the fixed apex. (b) *Ileo-colic*, the intussusception starts in the terminal ileum, the apex is just short of the ileo cæcal valve, and then proceeds into the colon. (c) *Ileo-ileo-cæcal*, the ileum prolapses into the ileum and passes to the ileo-cæcal valve, where it becomes wedged, pushes this before it, and then proceeds as an ileo-cæcal type with the valve as its apex.

The *caput cæci* type is a form of the ileo-cæcal in which the outer wall of the cæcum slips beyond the apex. On reduction it will present a characteristic dimpled appearance.

**Clinical Features.** Acute intussusception is met with most frequently in infants under the age of twelve months. The writer has seen it as early as two and a half months. The onset is characterised by a sudden fit of screaming, usually occurring in a male infant, previously in good health and spirits. The screaming is obviously due to severe abdominal pain. The infant kicks his legs and soon passes flatus. The bowels may act, and if the infant's stomach is not empty, he will vomit. The face will be seen to be pale and clammy, unlike the flushed and perspiring face of a child crying from a fit of temper. After the attack has passed off the infant lies in a state of exhaustion, giving an occasional moan; at varying intervals similar screaming fits succeed one another, and if the infant is fed during an interval he will vomit. At the end of four to eight hours the infant's napkin often will be found to contain some blood and mucus. Careful palpation of the

abdomen will show in the majority of cases that there is an emptiness in the right iliac fossa while a lump firm and sausage shaped may be palpated somewhere along the course of the colon. It may be felt in the ascending transverse or descending colons or in the pelvic colon or it may be felt like the os uteri on passing the finger through the anus. In late cases the intussusception may protrude from the anus.

**Diagnosis** This can be made from observation of the succession of screaming fits associated with vomiting (if nourishment has been given by mouth) together with the expulsion of flatus one or two motions and blood and mucus per anum and a tumour increasing in size and advancing in position. In cases of doubt an anæsthetic should be given when the whole abdomen can be explored by bimanual palpation. Even when the child is anæsthetised it may be difficult to identify an intussusception which lies in the hepatic or splenic flexures.

**Differential Diagnosis** (1) *Dysentery* In severe cases of dysentery the passage of blood and mucus may cause confusion in the diagnosis. Pain is less severe than in intussusception however and the stools are more frequent and contain fecal material while there is no abdominal tumour to be felt.

(2) *Rectal Prolapse* This condition is never associated with vomiting and severe colicky pains. If the protruding gut is reduced and the finger inserted into the rectum the intussusception will be found still within the bowel and the finger can be passed around it and between it and the bowel wall. The finger can also be passed along the side of the protruding bowel through the anus into the rectum. This cannot be done in a case of anal or rectal prolapse.

(3) *Henoch's Purpura* This disease is characterised by abdominal pain vomiting the passing of bloody motions and eruption of purpuric spots. It occurs at a later age than is common in intussusception. Rarely the two conditions co-exist.

(4) *Tuberculous mesenteric glands* may cause pain and a swelling in the abdomen. The onset of symptoms is more gradual and the condition is less acute.

A barium enema will definitely establish the diagnosis.

**Treatment** Immediate laparotomy with reduction of the intussusception should be performed.

If the gut is gangrenous resection must be performed but the mortality of such cases is nearly 100 per cent.

**After treatment** If shock is present the foot of the cot can be raised and rectal salines should be given. Sips of cold boiled water should be given frequently. In three or four hours if the child is breast fed breast feeding may be started small feeds being given at first. The same principles apply to bottle feeding. Older children can be given diluted milk or glucose and water. No purgatives should be given and if the bowels do not act by the second day a small rectal wash-out should be administered.

### Hirschsprung's Disease (Idiopathic Dilatation of the Colon)

This disorder is characterised by constipation associated with gradual enlargement of the colon to enormous proportions. The condition may commence early in the first year though it is seldom diagnosed at this age. It is much more common in boys than girls. The constipation is of a curious type. The bowels only open at long intervals (e.g. at weekly two weekly or even four weekly periods). At these times an offensive diarrhoea occurs for a number of days till the contents of the colon have been evacuated and then constipation again sets in. Drugs have no apparent effect. Much toxic absorption takes place from the bowel and the child's growth and development are retarded. He appears sallow and unhealthy, has a poor appetite vomits occasionally and sometimes complains of abdominal pain.

On examination the abdomen is found much enlarged. The colon can often be made out and peristaltic waves seen passing along it. Rectal examination reveals no obstruction and a ray following a barium enema though showing a greatly distended colon reveals no visible obstruction.

**Pathology** The colon is found at autopsy to be greatly dilated the greatest dilatation being at its lower end. Its muscle coat is thickened. No obstruction is found.

Numerous theories have been put forward to explain this curious phenomenon of dilatation without apparent obstruction. It is now generally considered to be a disorder of the sympathetic nervous system.

**Treatment** Medical treatment merely consists in clearing out the bowel by enemata and is purely palliative. Drugs are useless.

**Surgical Treatment** The literature contains a rapidly increasing number of cases of Hirschsprung's disease treated by sympathectomy operations. Although most of the early results are encouraging sufficient time has not yet elapsed



FIG. 4a.—Hirschsprung's disease in a boy of five and a half prior to sympathectomy.

to enable us to assess the permanent value of these operations.

The value of sympathectomy can be tested prior to operation by giving the patient a barium enema, turning him over on the left side and injecting a spinal anæsthetic in a dose sufficient to produce analgesia as high as the umbilicus.

If after this procedure the patient complains of a colicky pain, a wave of peristalsis is seen to pass along the colon and a quantity of barium is expelled from the rectum, it may be assumed that the previously inert bowel is contracting and

the operation of sympathectomy may be undertaken with advantage

The following notes were taken from a recent case of Hirschsprung's disease operated upon by the author. A boy five and a half years of age the mother stated that from infancy he had been



FIG. 45.—Hirschsprung's disease in same case fourteen months after operation.

constipated. The constipation gradually became more marked and during the past four years the bowels moved every ten days. Various purgatives had been given but it was noticed that the bowels acted equally well with and without them. For the past six months the child had been getting listless and easily tired. The abdomen showed marked prominence particularly of the epigastrium. Visible peristalsis could be seen mainly in a downward direction most marked in the epigastrium and umbilical region. The abdomen was very soft and hyperresonant. Skiagraphs of the barium enema examination are reproduced. These show marked dilatation from the transverse colon to the rectum.

Operation was performed on November 4th 1935. The technique advocated by Rankin and Learmonth was adopted which entailed division of the presacral nerve over the sacral promontory and stripping of its middle and lateral roots off the left common iliac vein and the bifurcation and anterior aspect of the aorta as high as the origin of the inferior mesenteric artery. The sympathetic fibres converging upon the origin of this vessel from above and from the sides were all divided and finally the proximal inch of the artery was completely bared so that all the sympathetic nerves running with it to the bowel were cut.

Some days after the operation the bowels acted and have continued to act daily and sometimes twice daily without aperient and the child's general condition has improved steadily.

See photographs Figs 45 and 46 and x rays Plates XIV and XV

### Spina Bifida

In early embryonic life the nervous system develops in the form of a groove the lateral folds of this uniting dorsally to form the neural tube. Should this process of development go wrong there is defective closure of the neural tube associated with a similar defect in the closure of its bony vertebral canal, hence the term spina bifida. The neural arches of the vertebrae unite in the mid line dorsally beginning in the dorsal region and extending up and down the column. The lumbo sacral and the cervical regions are the last to unite and it is here that the abnormal closures most frequently occur. The defect in closure allows a protrusion of membranes in a sac. At birth this sac may be merely an inconspicuous bulge or may vary in size to that of a translucent globular sessile bag as large as an orange. The sac enlarges during crying or coughing. If compressed the tension in the fontanelle will be increased.

There are certain degrees of severity of the condition which may be tabulated in terms of the neural structures underneath as follows —

(1) *Myelocoele* results from arrest of development at the time of the closure of the neural groove. The central canal of the cord opens upon skin surface. In the lumbo sacral region an elliptical moist vivid red area is seen. Myelocoele is the most common type of spina bifida with the exception of spina bifida occulta. Many of the infants suffering from the condition are still born and if the child is born alive it soon succumbs to infection of the cord and meninges.

(2) *Syringo myelocoele* consists of a sac caused by fluid distension of the central canal the cord forming a thin cyst wall

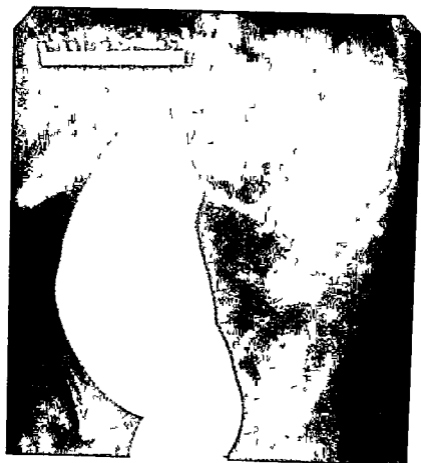
# PLATE XIV



## MEGACOLON

Barium enema. Extreme dilatation of pelvic and descending colon prior to sympathectomy.

# PLATE XV



## MECACOLON

Barium enema. Fourteen months after sympathectomy, there is still a good deal of dilatation of pelvic colon, but the upper part of the large intestine is almost normal in appearance.

The nerves pass outside the cyst This is the rarest type of spina bifida

(3) *Meningo-myelocoele* consists in a dilation of the membranes to form a cyst The normally developed spinal cord is displaced into the posterior subcutaneous surface of the sac and this structure is visible through the thin wall with its nerve roots stretching out laterally

(4) *Meningocele* consists of protrusion through the bony defect of a process of the membranes containing clear fluid which is sometimes under tension The skin may be either normal or greatly thinned out The communication between the sac and the spinal canal may be wide or very narrow

(5) *Spina bifida occulta* is due to failure of the neural arches to unite, but there is no protrusion of the membranes or cord One vertebra only may be affected in the lumbar region The defect may be felt as a depression and over it there is frequently a dimple and rarely a tuft of hair This condition may cause few symptoms in infancy and the deficiency is recognised usually when a radiograph has been taken for some other reason It may have disastrous results during adolescence and after

Rarely a defect is found in the vertebral bodies ventrally (i.e., anterior spina bifida) It occurs generally in the sacral region and gives no symptoms In later life a tumour may be found on pelvic examination

*Spina bifida cystica* may be associated with no other problem than that of the urgent necessity of preventing rupture of the sac and the resultant death of the infant from septic meningitis Frequently however it is associated particularly in meningo-myelocoele with paralysis of the lower extremities loss of reflexes and incontinence of the urine and feces The latter can be recognised in an infant by a patulous anal sphincter devoid of tone Hydrocephalus is a frequent concomitant

The frequent association of hydrocephalus with spina bifida has been observed for centuries but no explanation of this association has yet proved wholly acceptable Russell and Donald (1935) offered a mechanical explanation for the production of communicating hydrocephalus in a series of ten cases of meningo-myelocoele These observers showed that in all their cases there was a remarkable malformation of the hind brain In this a tongue of variable length consisting of cerebellar tissue and the greatly elongated medulla oblongata,

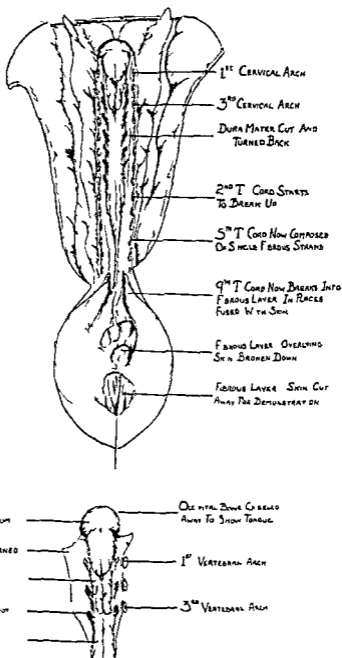


FIG. 4<sup>th</sup>—Drawing from post mortem specimen of meningo-myelocoele. The Arnold Chiari malformation and resection of cervical roots are represented.

protruded downwards into the spinal canal. It overlapped and compressed the upper segments of the cervical cord, distending the dural theca in this neighbourhood and filling the foramen



FIG. 48.—Meningocele.

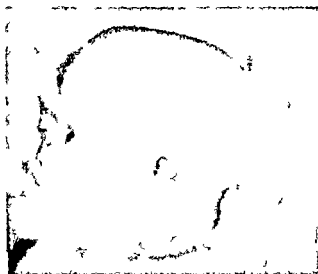


FIG. 49.—Hydrocephalus which developed after operation for meningocele.

magnum. The cavity of the fourth ventricle extended caudally into this tongue lying between the cerebellar and medullary components. The maximum diameter of the ventricle was usually within the vertebral canal at or below the level of the foramen magnum and here, too, at a still lower level was found

the choroid plexuses and the foramina with which they were associated. Another structural abnormality commonly found in association with this spina bifida is an abnormally small spinal cord from which the cervical roots run in a cephalic direction to reach their exits through the dura mater.

This curious maldevelopment was noticed first by Arnold (1894) and it was more fully described in the following year by Chiari and is described as the Arnold Chiari malformation.

It has been suggested that occlusion of the foramen magnum in cases of spina bifida may lead to a damming back of the cerebrospinal fluid in the ventricular system and hence to internal hydrocephalus.

**Treatment.** The treatment of spina bifida cystica depends largely upon its severity. A mere bulge in the lumbo-sacral region requires no interference, but the more common thin-walled cyst constitutes a grave menace to life. Rupture of the sac if the child struggles is inevitably followed by septic meningitis.

Infants with a severe form of spina bifida and associated hydrocephalus seldom survive either because of rupture of the sac or because of steady increase in the hydrocephalus. Treatment in the milder cases is aimed at protecting the sac till such time as surgical measures are most satisfactory. A pad of cotton wool built up in layers around the sac and secured by adhesive tape can be used. If the sac increases in size it may be tapped by a needle inserted obliquely through the surrounding normal skin.

Operative interference is rarely possible in cases of syngo myelocoele and meningo myelocoele as these cases usually exhibit paralysis of the limbs which cannot be cured or even improved. Cases of meningocele offer the most hopeful prognosis. If the skin over the sac is healthy it is advisable to postpone surgical plastic closure of the sac until after the first year because (1) surgical operations are tolerated better after this age, (2) should more grave defects such as paralysis of the legs or incontinence of the urine be associated their presence can be diagnosed with greater certainty after the early months of life. Their presence is of course a contra-indication to surgical interference. However if during the waiting period the skin shows signs of becoming gradually thinner and atrophic from increasing pressure operative treatment should be carried out. Operation entails exposure of the

are by means of an incision to one side of the mid line. The sac is opened on the opposite side to that of the skin incision. The sac is conserved and replaced in the vertebral canal without producing pressure on the cord. The spinal muscles are approximated over the bony defect and reinforced with flaps from the sheath of the erector spinae muscles. The wound should be well protected so as to prevent soiling. Till recently there has been no treatment for hydrocephalus developing after operation but now if the condition is thought to be due to a mechanical block at the foramen magnum by the Arnold-Chiari malformation decompression of the spinal cord at the foramen magnum may be attempted.

### Acute Osteomyelitis

**Ætiology** Acute osteomyelitis is a blood borne pyogenic process which originates in the cancellous structure of the metaphysis. The vast majority of cases occur between the ages of two and twelve years. Boys are more often affected than girls. The lower limb is usually affected. The commonest sites are the lower end of the femur and upper end of the tibia.

Other occasional sites are the lower ends of the tibia and fibula, upper end of humerus, radius and ulna, upper end of femur and the crest of the ilium. One bone only is usually involved at first but not infrequently secondary metastatic foci may appear later in other bones in cases which have not received early treatment.

The primary blood infection may arise from boils, septic abrasions, tonsillitis, otitis media, dental caries, pneumonia and umbilical sepsis. Injury of a trivial character is the most important general cause. Slight trauma causes metaphyseal capillary haemorrhages which afford excellent culture media for bacteria. The infecting organism is the staphylococcus aureus in about 90 per cent of cases. More rarely the streptococcus or the pneumococcus may be found.

**Pathology** At first the process is usually localised to the cancellous bone of the metaphysis but soon tends to spread rapidly, being accompanied by an inflammatory exudate and later followed by suppuration and generalised toxæmia. The rigid bony walls prevent immediate escape of the pus which soon comes to be under considerable tension producing severe pain.

Extension now usually occurs the most common route of extension being to the surface of the bone immediately superficial to the primary focus. The infection spreads to the subperiosteal plane and the subperiosteal exudate at first serous becomes purulent and strips the periosteum from the cortex over a varying length and circumference of the bone. The infection may spread centrally from the metaphysis into the medullary cavity and then reach the subperiosteal plane *via* the Haversian and Volkmann canals in the cortex.

The late Professor Starr of Toronto believed that the spread to the medulla occurs secondarily to the infection in the subperiosteal plane the route being inwards along the Haversian canals and not directly from the original focus in the metaphysis. The extent of the subperiosteal involvement varies occasionally the whole shaft is denuded. The infection does not spread easily towards the joint owing to the structure of the epiphyseal cartilage and the attachment of the periosteum to it. The periosteum in the region of the infection becomes congested and thickened. Finally suppuration may spread through the periosteum and extend to the surface through the soft tissues. When the periosteum has been elevated from the cortical bone the blood supply to the cortex is impaired and this will lead to necrosis of the bone and the formation of sequestra.

Whilst the above changes are occurring in the bone it is not unusual to find evidence of effusion in the neighbouring joint—sympathetic arthritis. At first the effusion is serous and sterile later it becomes purulent if the case is neglected. A virulent osteomyelitis may terminate fatally by rapidly producing an acute general septicæmia.

**Signs and Symptoms** The onset is sudden not infrequently accompanied by a rigor. There may be a history of slight injury such as a twist or sprain. (1) Intense localised pain in the metaphyseal region of a long bone generally near the knee. The pain may be referred to the joint but on careful examination there is absence of swelling deformity muscular spasm or restricted movement. (2) A point of extreme bony tenderness along the epiphyseal line can be detected. In the early stages no further local signs are present but the general signs of toxæmia are well marked. The child is flushed temperature high and the pulse frequency is raised. The tongue is dry, vomiting and delirium may occur.

In the later stages when the infection has spread to the subperiosteal plane a swelling appears with the classical signs of inflammation. The skin over the swelling becomes oedematous and fluctuating. The swelling is at first localised to one aspect of the limb. In lower femoral cases there is a fullness in the popliteal space with some flexion of the knee. In tibial cases the swelling may appear over the subcutaneous aspect of the bone or in some cases on the posterior aspect beneath the calf muscles. An effusion may be present in the neighbouring joint. In the later stages of the disease the general signs of toxæmia become less marked, the temperature is diminished, the pulse improves and the pain is diminished. This improvement is only temporary and relapse will follow with extension of the disease.

**Diagnosis** The diagnosis should be made as soon as possible before the infection has spread to the subperiosteal plane. The general signs of toxæmia associated with pain and tenderness in a long bone of a child should suggest the diagnosis of acute osteomyelitis. Radiographs are of no help in making the diagnosis in the early stages.

**Differential Diagnosis** (1) *Acute rheumatism* limited to one joint hardly ever occurs. Joint signs are present from the beginning. There is absence of bone tenderness.

(2) *Cellulitis* shows an infected abrasion, or history of an insect bite. The infection is confined to the soft tissues from the onset.

(3) *Acute pyogenic arthritis* shows a swelling which is confined to the joint. There is generalised spasm of all the muscles surrounding the joint.

(4) *Acute Tuberculous Arthritis* There are the primary signs of arthritis.

**Complications** (1) *Septicæmia* is by no means uncommon, but it is the experience of most surgeons that acute osteomyelitis is much less virulent than formerly. This is probably due to improvement in the health and nutrition of children. When septicæmia occurs the outlook is grave, and the majority of these patients die.

(2) *Secondary foci* may occur in any part of the body. These may require appropriate treatment. The outlook in these cases is also serious.

**Arthritis** A serous effusion into the neighbouring joint is not very common. It does not require any treatment. It is

usually absorbed and does not leave any ill effect. Septic arthritis is rare but when it does occur requires immediate treatment.

*Pericarditis and Endocarditis* These complications may arise quite early in the course of the disease and should always be kept in mind.

*Pneumonia and nephritis* are occasionally seen as complications.

**Prognosis** This is always grave depending upon the virulence of the organism, duration, symptoms and the resistance of the patient.

**Treatment.** Immediate operation should be performed. Cases operated upon within the first forty-eight hours yield the best results.

### Acute Arthritis of Infants

This is a rare condition which starts as an epiphysitis and then rapidly involves the joint. Its onset is abrupt and acute, the condition being associated usually with some other focal pyogenic infection (e.g. pneumonia). It is met with during the first two years of life. The commonest joint to be affected is the hip, more rarely the shoulder or other joint. Occasionally multiple lesions occur. The infecting organism is either the staphylococcus, the streptococcus or the pneumococcus.

As a rule the epiphysis is completely destroyed with resulting deformity due to shortening of the limb and subluxation of the joint. Ankylosis is rare.

**Symptoms** The child becomes restless, cries and resents being touched. His temperature is raised and pulse rapid and general appearance that of a very sick baby. On examination the limb involved is found to be held rigid, the joint is stiff, as the process extends the surrounding tissues become swollen and sometimes red.

**Diagnosis** in the early stages may be difficult but can be made easily when the swelling of the joint and coincident muscular spasm appear. The child tends to hold himself motionless and hence the condition must be diagnosed from scurvy. Osteomyelitis and acute poliomyelitis in the early stages have also to be borne in mind. In doubtful cases help may be obtained from the leucocyte count and x-ray of the bones. In acute arthritis the leucocyte count will be increased but the skiagraph may show no joint changes in the early

stages In scurvy the leucocyte count will be normal and the skiagraph will show subperiosteal hemorrhage In osteomyelitis the leucocyte count will be increased and the skiagraph will show the bony lesion, though in the early cases again the latter may be indistinct In polyomyelitis the diagnosis will be made by the absence of the above signs and the later appearance of paralysis of groups of muscles

**Prognosis** The condition is always a grave one with a high mortality and a frequency of deformity in those babies who recover

**Treatment** Arthrotomy should be performed and the joint drained by a tube down to the capsule The loose epiphysis should be removed The infant should be placed in an abduction frame, with the limb in abduction with extension This prevents subluxation of the joint, until the soft tissues have contracted Usually the functional result is poor

### Developmental Defects of the Rectum and Anus

Congenital defects of the rectum and anus are rare They may be classified as follows

(1) Imperforate conditions, (2) absence of the rectum or anus, or both, (3) opening of the rectum or anus in abnormal situations

*Imperforate anus or atresia ani* is undoubtedly the most common, and is observed most frequently at the ano rectal junction Its presence is to be explained most probably by a persistence of the cloacal membrane

*Complete absence of anus* is sometimes seen In such cases the rectum ends blindly in the vicinity of the anus, or may open elsewhere

*The rectum may be absent either in part or completely* It may end blindly at the upper border of the vagina or prostate due to absence of the post allantoic gut A fibrous cord can usually be made out, leading from the blind extremity of the rectum down to the anus, and is supposed to represent the shrivelled portion of the primitive rectum When the rectum opens into the bladder or into the deep urethra, the condition would appear to be due to an imperfect closure of the anterior and posterior parts of the cloaca

Abnormal openings may be found in the male in the region of the scrotum or penis, and in the female in the region of the

vulva. This is probably due to fusion of the margins of the cloacal fossa at the anal site.

**Signs Symptoms and Treatment** Very few cases are recognised until signs of intestinal obstruction develop or failure to pass meconium is noticed. Fæcal traces may be found in the urine or coming from the vagina in cases where a fistulous track connects the blind end of the rectum with the bladder urethra or vagina. If the fistula opens on the surface in the region of the scrotum or perineum a slight trace of meconium may be seen. Careful examination should be done under good light. If the anus is present a finger tip introduced gently will reveal the rectal occlusion. A definite bulging indicates that the rectum is only separated by a septum. The septum should be incised crucially and dilated with the finger. Absence of bulging shows that the rectum is imperfectly developed and the exact localisation of the depth of the bowel from the surface is determined by means of the air bubble when the infant is held upside down in front of the x ray tube. In this case an incision is made in the posterior part of the perineum and the dissection is carried out in the direction of the hollow of the sacrum. The blind end is drawn down sutured to the skin about the anus and opened.

If the bowel cannot be found and so brought down the only resort is left inguinal colostomy. This operation will also have to be performed in cases when the rectum communicates with the bladder or urethra.

In cases where an external fæcal fistula is present these outlets are usually unduly small and require dilatation. This is done by using a fine probe gently introduced so as not to make a false passage followed by small Hegar's dilators. A deliberate plastic operation can be planned at a later date.

**Other Types of Atresia** Atresia may occur in almost any part of the alimentary canal the commonest being the duodenum. Symptoms of progressive obstruction (e.g. vomiting constipation and distension) appear shortly after birth. In all cases as soon as the diagnosis is made a laparotomy should be done under local anaesthesia if possible and the necessary enterostomy operation attempted if the circumstances permit. Sufficient successes of gastro-enterostomy have now been reported in cases of duodenal atresia to warrant operation in every case.

## Phimosis

Phimosis is usually congenital. In a well marked case the prepuce is unusually long and its orifice quite small. In this type of case circumcision is most clearly indicated as the condition is often attended by difficulty with micturition which causes straining which in turn may occasion the development of a hernia or a rectal prolapse. Reflex disturbances too are common with this condition owing apparently to irritation beneath the prepuce.

In cases where the prepuce is not unduly long but the orifice is small the condition can be treated in early infancy by stretching the prepuce forcibly until it can be retracted over the glans. An anæsthetic is rarely necessary. Another simple method of treating these cases is the dorsal slit method.

Usually a general anæsthetic is given using ether dropped on to a Skinner's mask. The child's buttocks are placed on a small sand bag and the chest and upper part of the abdomen are covered with a small blanket. The theatre or room should be suitably warmed. The penis and scrotum are painted with tincture of iodine and sterilised towels are arranged so as to leave the penis exposed. The dorsal portion of the prepuce is picked up with two small hæmostatic forceps placed about  $\frac{1}{2}$  inch apart one on either side of the mid line. Adhesions between the prepuce and glans on its dorsal aspect are broken down by means of a probe or a narrow blunt dissector. Avoiding the dorsal vein which can be seen a medial dorsal incision is made with blunt pointed scissors between the forceps half way from the meatus to the corona. After the dorsal incision has been made the prepuce is folded back and masses of smegma if present are removed with a dry swab. Then the mucous membrane is incised completely up to the corona. Avoid injuring the *frænum* otherwise bleeding will occur at this point. The artery forceps are removed and any small bleeding points on the cut edges can be ligated with very fine catgut if necessary. A narrow strip of sterile gauze is wrapped around the penis as so to keep the prepuce drawn back over the glans. The redundant tissue gradually shrinks and the part eventually resumes a normal appearance.

In cases where the prepuce is long and too much redundant tissue would be left after the dorsal slit operation a circumcision should be done.

**The Operation.** A general anæsthetic is given by means of ether dropped on to a Skinner's mask. The parts having been cleansed and disinfected in the usual way the preputial orifice is dilated with artery forceps the prepuce peeled back and smegma if present removed with a dry swab. The prepuce is then brought forwards

again and seized on its ventral aspect in the mid line with artery forceps. The forceps are applied so that the points are close to the frænum. The skin and mucous membrane are divided completely just proximal to the point of the forceps so as to divide the frænum. With slight traction on the forceps the preputial integument, including the mucous membrane is divided all round by successive snips of sharp scissors on alternate sides. The line of section on each side inclines downwards and backwards slightly and on the dorsum runs transversely just in front of the corona of the glans. The small frænal artery and any other bleeding points are seized with fine pointed hæmostatic forceps.

The first suture is introduced through the divided frænum and the adjoining integument. It consists of fine catgut and in introducing it an attempt should be made to under run the frænal artery. The ends of this suture are cut and left long. The second suture is introduced behind the centre of the corona through the skin and mucous membrane. It is also cut and the ends left long. By drawing these sutures apart the introduction of the other sutures on each side are made easy as the skin and mucous membrane edges approximate. Finally all the sutures are cut short a zinc ointment dressing applied around the end of the penis and renewed every day until healing occurs.

### Paraphimosis

A tight prepuce has retracted over the glans and cannot be returned. The glans becomes swollen and cedematous.

**Treatment.** A general anæsthetic is given and the constricting band divided with a scalpel on the dorsal aspect. Reduction will then be successful. A "dorsal slit" or circumcision can be performed later.

## CHAPTER XXIX

HENRY STOKES

### ABDOMINAL HERNIAS UNDESCENDED TESTES AND HYDROCELE

(Hernias Umbilical — Inguinal — Strangulation — Internal Undescended Testes — Hydrocele)

#### Abdominal Hernia

A HERNIA is a protrusion of a viscus from its normal cavity through an abnormal opening it may be external or internal

#### External Hernia

A Umbilical Umbilical hernia is very frequently met with during the first six months of life The opening may be in the linea alba above the umbilicus or the latter may remain patent The condition may be present at birth Hence any swelling along the cord must be carefully reduced before the cord is ligatured The hernia is covered by skin and peritoneum and contains small intestine and sometimes omentum As a rule it is easily reduced the process being accompanied by a gurgle

Umbilical hernias never strangulate and show a marked tendency to close

Treatment Palliative measures as a rule make little difference but as mothers often become worried at the protrusion it is wise to reduce the hernia and keep it in place by strapping Rubber belts are unnecessary and less satisfactory If the condition has failed to clear up by the time the child is three years operations should be performed The sac is exposed the contents reduced the excess of sac cut away and the opening sewn up

B Inguinal Hernia Inguinal hernia is the result of a faulty obliteration of the funicular process it may be unilateral or bilateral if the former it is more commonly seen on the right side The condition is sometimes associated with undescended testicle or hydrocele Inguinal hernias contain small or large

intestine in infants the omentum is too short to reach the inguinal canal

**Diagnosis** These hernia are variable tending to come down when the baby cries or strains and reducing themselves in the intervals. If seen when the sac is full the diagnosis presents no difficulty but if the baby is brought to the doctor in the interval when the sac's contents have returned to the abdominal cavity the diagnosis may be difficult. The history of 'a swelling' given by the mother is often unreliable and the size of the inguinal ring a poor guide. Observation is the best course to take in these circumstances as strangulation seldom occurs in the baby.

Hydrocele of the cord is sometimes mistaken for a hernia. It may be slowly reducible but this is never accompanied by a gurgle.

The condition of undescended testicle is always associated with a potential hernial opening.

**Treatment** There is a marked tendency here also for spontaneous cure though this always leaves a potential hernial sac which may give rise in later life to a sudden rupture. Hence some authorities state that all inguinal hernias seen in infancy should be operated upon. Temporarily at least a great many of these hernias will respond to non-operative treatment however the usual method being to reduce the hernia and fit the baby with a rubber truss.

The choice of time for surgical treatment depends upon the age and general health of the baby. Many cases of inguinal hernia disappear during the first six months of life but spontaneous cure does not occur after this age. Hence if a hernia persists after six months of age it should be operated upon as soon as the child's general condition is suitable. A baby should never be given a general anaesthetic unless it is in good health. Many of the infants seen in hospital practice with inguinal hernias are sickly and malnourished. It is always wise to correct the feeding and get the baby fit before sending him to the surgeon for operation. On the other hand the hernia may be large and the mother unable to manage the truss hence the problem of the most suitable moment for operation is often a difficult one and occasionally a choice between two evils has to be made.

If the hernia is associated with undescended testicle operation should be postponed till the testicle has reached the

scrotum, as the hernia will aid the process of descent by pushing the testicle down in front of it (Irreducible hernias always contain large intestine and, when painful, sometimes the appendix) If the hernia is large irreducible and tender it should be operated upon at once whatever the age of the child, as strangulation may occur

*Strangulation* may occur in babies from three weeks of age The baby cries draws up the legs and appears in pain On examination a painful irreducible, tense swollen hernia is found Vomiting occurs early and if the condition is left unrelieved may become faecal The diagnosis is by no means easy and the condition has to be differentiated from torsion of the cord associated with undescended testicle or prolapsed ovary or acute appendicitis in the sac In cases of torsion of the cord with undescended testicle the absence of the testicle from the scrotum and a tender swelling in the inguinal canal on that side will usually give the necessary clue In appendicitis in the canal the temperature will be raised early, and later there will be swelling and oedema of the parts

Sometimes the diagnosis will only be made for certain at operation The treatment of strangulated hernia is early operation, whatever the age of the baby As a rule after dividing the obstruction the contents of the sac can be returned to the abdominal cavity Occasionally in late cases gangrene will be found and resection must be attempted

### Internal Hernias

Internal hernias are nearly always due to some congenital abnormality, e.g., incomplete omentum, containing holes through which the gut may pass, or incomplete diaphragm The latter may lead to large internal herniation of abdominal contents into the thoracic cavity which may or may not lead later to strangulation

The diagnosis of these cases is often difficult or impossible if acute obstruction has supervened Where the condition has become chronic, however, a barium meal and a skiagram is the best way to obtain definite information as to the exact state of affairs Operation is usually unsatisfactory in these conditions, but should be attempted if strangulation occurs or threatens

## UNDESCENDED TESTES

In this condition the testes may lie in the abdomen or in the inguinal canal if in the latter as we have seen the condition is associated with a potential hernia

The scope of this work deals primarily with babies during the first year of life. During this period operation for undescended testicle is never justifiable. The majority of undescended testes come down on their own the condition being very rarely met with in adult life. If the condition is persistent modern treatment with gonadotropic hormone may be attempted later and if this fails operation must be considered before the boy reaches puberty.

## HYDROCELE

The condition is very common during the first year of life particularly in new born infants. It consists of an effusion into some portion of the peritoneal pouch which has been brought down the inguinal canal its pathology resembling that of inguinal hernia. There are a number of varieties it may be confined to the tunica vaginalis or a portion of the cord may stretch down the canal from the peritoneal cavity for a variable distance.

The diagnosis has to be made from inguinal hernia. In the isolated types when in the tunica vaginalis or when it appears like a cyst on the cord it is not reducible is clearly transilluminable and remains constant in size and shape for a considerable period of time. The type which communicates with the abdominal cavity may be slowly reducible by slow pressure in contrast to the ordinary reducible inguinal hernia which goes back all together with a gurgle. Occasionally a hydrocele and an inguinal hernia occur together.

The prognosis is good most patients making a spontaneous recovery within a few months without operative interference being necessary.

If the hydrocele is large or if it fails to clear up it should be tapped and if it recurs the sac should be injected with varixol (quinine-urethane).

## CHAPTER XXX

WILLIAM DOOLIN

### CLEFT LIP AND CLEFT PALATE

(Primary Effect of Cleft upon the Infant    Type of Cleft    Nature of the Surgical Problem )

ON an average estimate, about one out of every thousand children born comes into the world with a congenital defect of lip or palate, or both. Peron \* (1929) reviewed the records of 100,000 children born in the Paris maternities, and found the incidence to be 1/942. In the United States, Warren Davis, † in a similar investigation undertaken in 1924, had found the incidence to be 1/915. Peron further estimated that the primary infantile mortality in such congenital defectives was extraordinarily high, 22 per cent. of them dying within a few days of birth.

The effective correction of these congenital defects belongs to a particularly difficult branch of plastic surgery. It is questionable, even, if the operation designed to restore a highly complicated musculature to its normal functioning capacity should be attempted by the average general surgeon. The removal of a fibroid uterus or the resection of a loop of bowel arc, by comparison, surgical procedures much more easily accomplished than is the plastic repair of a cleft palate. The late Sir James Berry, with all the authority of his unrivalled experience of this particular field of surgery, has left on record the warning to the general surgeon —

The operator who has had little experience of this branch of surgery, and who finds himself called upon to do one of these operations, will do well to follow the old-fashioned plan of postponing the operation to a much later period. This is better than running the risk of running a child for life by spoiling its palate and possibly leaving it in such a condition that no other surgeon has any chance of remedying the condition. We have all seen examples of this, and we shall doubtless all agree that the operations we have to

\* *Thèse de Paris* 1929

† *Ann Surg*, 1924, 11, 363

do upon palates that have already been operated upon without success are more difficult to perform and are usually less successful in their results (*Proc Roy Soc Med*, 1927) \*

In a manual such as this, whose text is addressed to the senior student and the general practitioner, detailed consideration of embryology or of operative technique such as are still debatable points for the plastic surgeon, would be out of place. The majority of practitioners see but few of these cases in their individual practice. When the particular example is placed before him, it is the family doctor who is called upon in the first instance to find an answer to the questions put by the disappointed and anxious parents. It is in the effort to assist him to answer such questions that this chapter has been written.

### Primary Effect of Cleft Upon the Infant

The immediate post natal mortality as estimated by Peron in the Paris maternities is much too high in our experience. Whilst accurate figures for comparison from the Dublin maternities are not forthcoming, an approximate estimate of even 5 per cent. would be regarded by the latter authorities as high.

The primary difficulty in infants with congenital clefts of lip and palate is that of feeding. Some babies with lip cleft but palate intact can be easily breast fed. Few infants with a complete cleft of lip and palate have the power to suckle satisfactorily. In private practice the skilled maternity nurse can help to maintain the breast milk, express it, and feed the infant with the mother's milk either from a bottle with specially modified teat or by spoon or pipette. Wherever feasible it is advisable that the infant should be breast fed, but in those cases in which from physiological or economic reasons breast feeding is impracticable other methods must be adopted. The essential thing is that the child should show an average weekly increase in weight of about four ounces.

### Type of Cleft

The cleft may involve the lip alone, the palate alone, or lip and palate combined. Elaborate or complicated classifications

\* Cf Gainer (*Brit Journ Surg* 1913-14: 189). It seems that a special interest—and perhaps also a special capability—is required to make an other wise excellent surgeon into a successful operator in cases of cleft palate.

are of interest only from the ætiological or operative standpoints and will not be reproduced here. A cleft lip "jumps to the eye" of the least skilled observer, the family physician will need to note accurately whether it be unilateral or bilateral, whether it be *simple* or *incomplete* (i.e., involving the lip alone, with a fully formed nostril above the cleft) or *total* or *complete* (i.e., the cleft in the lip extends into the nostril above, which, as a result, is also deformed). These salient points noted, the palate should be examined for co-existent cleft.

The most frequent variety in the writer's experience has been the *total unilateral cleft*, involving lip, alveolar ridge (gum margin) and palate, in which the fissure extends from the lip in front to the naso-pharynx behind. Representative figures as to the relative frequency of the several types may be cited from an analysis of 500 consecutive cases treated by Veau and his assistant, Plessier at the Enfants Assistés, Paris.

For *cleft lip*, Plessier's\* analysis of their material gave the following percentages —

Unilateral, incomplete	33 per cent
Unilateral, complete	48    ,
Bilateral, incomplete	7     ,,
Bilateral, complete	12     ,,

The unilateral lip cleft, it will be seen, is the variety most commonly encountered, its relative incidence, either as complete or incomplete, being four out of every five cases (81 per cent.)

For *cleft palate* Veau† has differentiated his cases as follows —

Simple cleft of soft palate	20.8 per cent
Simple cleft of both soft and hard palate, alveolus normal	30.8    ,,
Unilateral cleft involving soft and hard palate, alveolus and lip	38.8    ,,
Bilateral cleft of palate, alveolus and lip	9.6     ,,

#### Nature of the Surgical Problem

To avoid painful misunderstandings and to eliminate possible

\* Plessier, "Bec de Lièvre unilatéral" Masson, Paris, 1931.

† Veau, "La Division Palatine" Masson, Paris, 1931.

sources of disappointment later the essential nature of the surgical problem should be made clear to the parents at the outset. In the child with an *incomplete cleft lip* the rational basis for operation is the closure of a divided oral sphincter the orbicularis oris musculature. Cosmesis is of definite but secondary importance. In cases of *complete cleft lip* the operator in addition to the closure of the oral sphincter aims at the reconstruction of the nostril with closure of the nasal floor both of these objectives should be attained at the one sitting. The closure of a *cleft palate* is usually deferred to a much later stage. The problem here is fundamentally different. In the cleft palate the surgeon has to deal with two distinct tissues. (a) The *hard palate*. He has here a rigid bony structure presenting a fixed gap. The problem is to find the material with which to bridge the gap. muco-periosteal flaps must be mobilised their coaptation and vitality assured. (b) The *soft palate*. Its structure is essentially muscular therefore mobile elastic with a constantly varying gap. Here there is plenty of tissue available to close the gap. the problem is to preserve its elasticity securing a solidly united muscular veil of sufficient depth and mobility to allow of adequate closure of the naso-pharynx. From the functional viewpoint—the subsequent attainment by the growing child of correct methods of speech—the physiological action of the soft palate is all important. The functional activity of the soft palate would appear to be greatly enhanced by the artificial narrowing of the naso-pharynx (pharyngoplasty) devised by Wardill\* of Newcastle-on-Tyne. the writer's experience of this procedure however is of too limited a nature to justify any expression of opinion as to its efficacy.

When should closure of the cleft be undertaken?

In general closure of the cleft lip may be undertaken as soon as the state of nutrition and the general health of the child will permit. Any child of 9 lb. and upwards if otherwise healthy is fully fit to undergo the strain of the operation. The weight of the child and its general state of well being are better criteria in the writer's opinion than any arbitrary decisions based on age. The average age at which the operation is undertaken is between the sixth and twelfth weeks. If there is no associated cleft in the palate and the child is thriving closure may be effected earlier than the sixth week to permit of breast

\* *Brit Journ Surg* 1938 xvi 47

feeding The operative risk of closure of a simple lip cleft is practically nil

Certain pre-operative requirements are essential The child *must* be in the hospital or nursing home for at least one week prior to operation Such an observation period is necessary to ensure that the child is not losing weight, has not been in recent contact with any case of infectious disease, and is free from any catarrhal affection of the respiratory or intestinal tracts (Broncho-pneumonia and gastro-enteritis are two of the most common causes of post-operative mortality in infants) Any trace of nasal discharge is a definite contra indication to operation the risk of infection of the suture line, with secondary disunion, far outweighs the inconvenience of postponement of the operation

If the infant is not thriving, operation should be postponed without hesitation The closure of a cleft lip is never an operation of urgency

What is to be attained by operation ?

Many unsatisfactory results of operation for cleft lip have been due to "not trusting to nature" In unilateral simple clefts, the primary aim of the operator should be the functional restoration of the divided oral sphincter (Kilner) Complicated zig zag flaps, aiming at an immediate cosmetic effect not infrequently produce the most disappointing results The simplest three layered suture of skin muscle and mucosa may not give the instantaneous improvement to the child's appearance desired by the mother, who fails to realise the possibilities of normal evolution, but the sound suture line which may look so disappointing within a few weeks of operation more often than not develops into a very satisfactory lip within a year Minor cosmetic readjustments are best deferred till adolescence

In cases of complete unilateral cleft lip the writer employs the operation devised by Veru, in which, by the co aptation of flaps cut from the mucous lining of the vomerine septum, the lateral nasal wall and the hard palate, the floor of the nose is first reconstructed, the nostril is then sutured as symmetrically as possible, and the closure of the lip defect is the terminal stage of the operation Closure of the soft palate is deferred to a second operation about a year later The sound muscular union of the orbicularis oris will generally succeed in moulding back into relatively correct position any unsightly projection of the pre maxillary element.

Cases of bilateral cleft of the lip with marked protuberance of the pre maxillary element are the least frequent—and the most difficult. In such cases the most satisfactory procedure is the three stage operation recommended by Veau—the larger of the two lip and nostril defects is closed as if the cleft were a unilateral one—two to four months later the remaining lip nostril gap is closed—the palatal closure is undertaken a year or so later. The simultaneous closure of both nostrils in cases of bilateral cleft lip has in the writer's hands proved a failure. Excision of the pre maxillary element is mentioned only to be condemned in the strongest terms—it is contrary to every principle of surgical anatomy and develops into the most appalling cosmetic deformity.

The criterion of a well executed operation for cleft lip is the development of a thick well muscled lip with a smooth unbroken muco cutaneous margin and approximately symmetrical nostrils. No perfect result of a cleft lip operation has yet been attained but the most disfiguring deformity can be reduced to minimal proportions by the painstaking surgeon.

### Closure of the Palatal Defect

The child whose lip has been successfully sutured but whose palatal cleft yet remains will be hard to rear. Chronic undernourishment is the main difficulty—with a complete cleft of the palate sucking either from breast or bottle is virtually impossible—not a few such patients of the hospital class succumb within the year from the ignorance or incapacity of those in charge of them. The hardier and the better-cared for survive and in them the question of the best time at which to undertake the closure of the palate has to be considered. The aim of the operator we have seen is to secure a closed partition between the buccal and nasal chambers restoring the soft palate in particular to such a condition that it can shut off the naso pharynx. What are the prospects of success? What are the risks of failure?

A dispassionate survey of the relevant European and American literature would show that the prospect of a complete anatomical closure of the cleft palate as the result of a single operation may be held out in two cases out of three. Depending on the operative procedure adopted in about one third of the cases two three or even four operative interventions may be

necessary, in these latter the functional results are disappointing in the extreme. In the vast majority of these recorded cases the method of closure has been the "classical" procedure of Langenbeck or one of its modifications. While this is not the place in which to enter on a discussion as to the relative merits of various operations the writer is definitely convinced of the general superiority of the technique so admirably worked out by Veau. The essential step is to secure a long mobile soft palate. By suturing the nasal and the buccal mucosæ in separate layers and being careful to spare the palatal musculature as much as possible the operator effects the closest possible restoration of the palate to its normal state.

There is a definite operative risk. Veau the master surgeon, had 19 deaths in 500 cases an operative mortality of approximately 4 per cent. The primary lethal factor is the age of the child. Analysed by years Veau's mortality was as follows —

In the first year	9.4 per cent
second year	5.7
third	2.7
„ fourth	1.8

The decreasing fatality rate with growth is a strong argument in favour of postponing the operation till the fourth year. But if one waits till then the chance of a good phonetic result is seriously diminished. Wardill has shown\* that speech defects are noticeable at or before the third year and once formed, are very difficult to eradicate. If the operation is undertaken before the end of the second year the prospects of normal speech are in the region of 70 per cent. One has to balance that prospect against a possible operative risk of 6 per cent. In the fourth year with an operative risk of less than 2 per cent the prospect of good speech is less than 25 per cent. (Here probably is the major field for the supplementary operation of pharyngoplasty as recommended by Wardill†) The family doctor asked by the parents. When should we have it done? is faced with a serious responsibility. Is it for him to make the decision? In the writer's view his role should rather be to instruct the parents placing the advantages and the risks of operation before them that they may take the responsibility. The surgeon thinking of the functional result will wish to

\* *Brit Jour Surg* 1933 xx 347

† *Lancet* 1930 i 143a

undertake the early operation it is for the parents to give him their authority to proceed

In expert hands the most favourable age for closure of the cleft palate is between the tenth and the twentieth months. To operate earlier is to run the risk of an unnecessary fatality; postponement to a later date will prejudice the prospect of a good functional result.

### Pre-operative Precautions

That the child be in vigorous health is a *sine qua non*. The closure of a palatal defect is a major operation and one of severity. One must be particularly careful to exclude the possibility of any febrile condition *e.g.* measles whooping cough scarlatina etc. For this reason a week's pre-operative observation in hospital is not a day too long. If the child is losing weight if he is coughing or running from the nose he should be returned to his family until any such contra-indication has disappeared. This happens more often in a children's hospital than in an adult service; it is sometimes difficult and has upon occasion led to disappointment with the parents but a strict adherence to this rule will prevent many operative disappointments.

Curiously enough fat babies—the type so favoured by patent food advertisers—withstand these operations much less well than thin ones.

Certain authorities from bitter experience recommend x-ray investigation of the thymus in all cases before operation. So far the writer has had no thymic deaths. In one case of sudden death on the table post-operative x-ray and post-mortem examination failed to reveal any thymic enlargement. As a warning signal of the existence of an enlarged thymus the nurse on duty should be instructed to be on the watch for any abnormality of breathing during the pre-operative week.

The removal of adenoid vegetations from the naso-pharynx prior to operation is an unnecessary complication. The writer has several times closed a palate leaving pronounced adenoids *in situ* without any ill-effect upon the post-operative course. So too with simple tonsillar hypertrophy. Active infection of the tonsils however will definitely indicate postponement of the operation.

At the age at which most of our cases come for operation the

question of dental caries does not arise. In the exceptional older child it would be well to have any decaying teeth attended to by a competent dentist.

The consideration of choice of anæsthesia, technicalities of operation, instruments, and the immediate after care of the operated infant belong more properly to text books of operative surgery. The subject of mortality having been alluded to earlier, the actual causes of such mortality may be briefly considered. These are chiefly two: post-operative broncho pneumonia and hyperpyrexia. After a properly executed closure of a palatal cleft, death from hæmorrhage should never occur. (The writer had one alarming case of secondary hæmorrhage in a boy of seven years following a classical Langenbeck operation, while the outcome happily was not fatal; the experience was such as to lead him to abandon this method of procedure.)

Deaths from broncho pneumonia are usually late fatalities; i.e. they do not occur within the first few days of operation. Owing to the skilful administration of endotracheal gas oxygen anæsthesia, no case of broncho pneumonia has occurred in our cases. The syndrome of pallor and hyperpyrexia has been met with by the writer in three instances, two with fatal results. The pathology of this curious development, unknown in older children, is wholly obscure. On the operating table the child becomes suddenly blanched for no obvious reason; no coincident hæmorrhage has occurred to account for the pallor. After restorative measures have been applied (one of the writer's cases died at this stage) the child is brought back to bed in a state closely resembling that of ordinary surgical shock. Within four to six hours mild convulsive movements of no special character may be observed by the nurse in attendance; these are associated with a beginning rise of temperature. The temperature continues to rise, reaching up to 104° F. within six hours; wet packs and cold enemata may reduce the temperature, but should these measures fail the temperature continues to rise, and death supervenes in syncope, usually within twelve to fifteen hours after operation. All the recorded cases have occurred in infants under two years of age.

### Results

Failure to secure primary union of the sutured cleft is the

result of faulty technique. The more experienced the operator the fewer cases of total or partial disunion will he have. But a sound anatomical union does not always produce satisfactory functional results. If the defect has been closed before the child has developed defective habits of speech and if the palatal musculature has not been interfered with the child should grow up without any phonetic disturbance. In private practice special speech training methods will obtain excellent results as time goes on but in children of the hospital class the elocutionary training available leaves much to be desired.

As simple tests of the functional result of the operation the ability of the child to snort to blow out a balloon or blow soap bubbles will clearly demonstrate the degree of functional closure of the naso-pharynx.

## CHAPTER XXXI

F J HENRY

### BURNS AND SCALDS

BURNS and scalds are extremely common in young children. Hospital experience shows that they are most frequently caused in one of two ways. Either the child's clothes become ignited through undue proximity to an unguarded fire, gas or electric stove, or else the child upsets the boiling contents of a kettle or saucepan over himself. The prophylactic measures in either case are obvious.

Although in recent years the prognosis of these injuries has been enormously improved by the introduction of treatment by tannic acid, their mortality nevertheless is still very formidable. In the vast majority of fatal cases death takes place within the first few days and is due to shock and toxæmia following the absorption of poisonous substances elaborated in the burnt tissues, while a few patients having survived the stage of initial shock perish some weeks later from sepsis. The prognosis of a scald is very much more favourable than that of a burn of similar size.

The therapeutic indications therefore in dealing with a severe burn are to treat shock, to reduce toxæmia and to endeavour to prevent infection, and treatment therefore resolves itself into the application of general anti-shock measures and the local treatment of the actual burn.

**Treatment of Shock.** In the first place administer a dose of opium suitable to the age of the patient. The most satisfactory preparation perhaps is tinct. opii ℥  $\frac{1}{4}$  in the first three months of life, ℥  $\frac{3}{4}$  in the second three months and after that ℥ 2 for each year of age. Then apply warmth by means of blankets, hot bottles or if possible by placing a radiant heat cradle over the patient. If stickily singed clothing has to be removed it is best done while the child is immersed in a warm bath. Finally give plenty of warm fluids to drink and in severe cases administer saline by rectal, subcutaneous or intravenous routes.

**Local Treatment of the Burn** Before any dressing is applied it is absolutely essential that the burnt area should be thoroughly cleansed. This is naturally a painful operation and should not therefore be undertaken until the patient is well under the effects of opium. Indeed in some cases gas and oxygen anæsthesia is necessary in addition but no other inhalation anæsthetic is advisable as these patients are very prone to pneumonia. All blisters must be opened and loose or dead skin cut away and the whole surface gently but thoroughly cleaned first with warm water and soap and then with ether. When this toilet and debridement have been satisfactorily completed the area is ready to be dressed.

It can be stated quite definitely that the only dressing which one is justified in applying to a recent burn is some preparation of tannic acid and in cases of emergency where this substance is not procurable quite a satisfactory substitute is available in the form of cold tea in the strength used for drinking. Oily dressings or ointments of all forms are to be particularly avoided.

The advantages of tannic acid are as follows: first it coagulates the burnt tissues and fixes the poisonous substances which these contain thereby reducing toxic absorption to a minimum. Secondly the coagulated tissue forms a leathery protection for the burnt area under which healing proceeds thus completely relieving pain and obviating the necessity for the ordeal of frequent dressing a most important matter in children.

Tannic acid may be applied in three ways. (1) In the form of tannafax jelly which is liberally smeared over the burn and the area then covered with several layers of lint or gauze. This is an easy and convenient method in small burns but does not give such good results in extensive cases as either of the two methods to be described.

(2) In the form of a 2 per cent solution containing a little perchloride of mercury applied by means of a spray. The solution should be freshly prepared and is conveniently made by dissolving in 2 oz of warm water a powder consisting of g 17½ of tannic acid and g ½ of perchloride. This solution is sprayed on to the burn every half hour until the whole surface is covered with a dark brown coagulum. As many as twelve applications may be necessary. The area is left uncovered but protected from the bedclothes by a cradle, if it involves a limb this should be immobilised on a suitable splint.

(3) As a compress consisting of several layers of sterile gauze or lint soaked in the above mentioned solution applied to the burnt area and slowly dried. This method was devised by Mitchner to whose writings the reader desirous of further detail is referred and has the advantage of being extremely simple and easily applicable to the conditions of general practice.

Certain precautions must be observed in using tannic acid. In the first place it must only be applied to recent burns in which severe infection has not yet become established. If its application be delayed for more than seventy two hours after the burn has been sustained there is a grave danger of imprisoning inflammatory exudates under the coagulum. In the second place the coagulum once formed must be kept absolutely dry and on no account whatever should a moist dressing be applied to it. Water will liberate the precipitated toxins and severe and possibly fatal toxæmia will ensue. Mild infection occurring under the edges of the coagulum need occasion no alarm but if any gross collection of pus should accumulate a window should be cut in the coagulum to allow its escape.

## CHAPTER XXVII

W S HAUGHTON with L B SOMERVILLE LARCF

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### ORTHOPÆDIC CONDITIONS

#### Introduction

ORTHOPÆDIC surgery as applied to infants is the surgery of deformities

Every child should be subjected to a short routine orthopædic examination as soon as possible after his birth. Most of the deformities with which a child can be born are comparatively easy to treat in the early years of life but when neglected the prolonged stay in hospital and the expensive splints and equipment required in the treatment of advanced deformities constitute a considerable drain on the private purse or the public exchequer.

Anyone who has worked in a public orthopædic hospital will have been struck by the advanced age at which patients are referred for treatment with severe crippling deformities which have been present since birth. The routine school examination now being carried out has done much to bring these cases to hospital earlier for treatment but the school age is too late to secure the best results in many conditions. The training of nurses and midwives in the recognition of orthopædic conditions is of the very greatest help in this respect.

It must be fully understood that the treatment these congenital deformities require differs considerably from that required by most surgical conditions. An operation in these cases does not cure. A deformity is made up of abnormalities in many structures. Ligaments muscles nerves and arteries may be either too long or too short stretched or contracted. Bones may be abnormally shaped misplaced or entirely absent.

The essential requirement of treatment in such cases is that by gentle and gradual means such structures may slowly be encouraged to assume normal shapes positions and lengths.

The employment of sudden or great force with the object of

obtaining quick correction of a deformity, or of overcoming powerful resistance in shortened structures, becomes less and less necessary as cases are referred earlier for treatment. Forceful treatment always produces a corresponding amount of trauma which militates against the best functional result, whereas correction by gradual and gentle means producing a steady pressure upon growing structures and a negligible amount of trauma will always give a better result.

The routine examination of the child should include examination of both feet both legs spine the arms and hands neck and head. Measurements of the legs should be taken. This examination need not take more than five minutes. It should be repeated as soon as the child is able to walk as some abnormalities do not manifest themselves until then.

## CONGENITAL DEFORMITIES

These are of two types —

- (1) Primary
- (2) Secondary

(1) *Primary congenital deformities* are inherent defects of the fertilised ovum which influence the development of the embryo spontaneously without outside cause.

(2) *Secondary congenital defects* are those where the foetus is normally formed but owing to some extraneous cause abnormalities arise at some later stage.\*

### Feet

The congenital deformities found in feet in the new born are —

(1) **Congenital Club Foot or Talipes Equino Varus.** This consists of a combination of various deformities.

- (a) An *equinus* or *dropped foot* deformity, occurring at the ankle joint.
- (b) A *tarus* or *inversion* deformity of the whole foot, occurring at the sub astragaloid joint i.e. the joint between the talus (astragalus) and calcaneus (os calcis).
- (c) An *adduction* deformity with *inversion* and *plantar flexion* of the fore foot occurring at the mid tarsal

\* Jones and Lovett



FIG 50 — Double club feet Front view



FIG 51 — Double club feet Back view



FIG 52 — Talipes varus



FIG 53 — Congenital flat foot  
The prominence caused by the head of the talus can easily be seen



FIG 54 — Congenital flat foot with valgus deformity

joint, i.e., the joint between the talus and navicular (scaphoid) medially, and the calcaneus and cuboid laterally

(2) *Talipes Varus* An inversion at the sub astragaloid joint

(3) *Talipes Valgus* An eversion at the sub astragaloid joint

(4) *Talipes Calcaneus* An abnormal dorsiflexion of the foot at the ankle joint so that the upper surface of the foot can touch the front of the tibia

(5) *Metatarsus Varus* Adduction of the fore foot at the mid tarsal joint

(6) *Metatarsus Valgus* Abduction of the fore foot at the mid tarsal joint In order to recognise these deformities every foot must be palpated and put through its full range of movements

### Club Foot

This is the commonest congenital deformity of the foot occurring in 0.1 per cent of all children. It is about twice as common in males as females. It may be either primary or secondary in type—the latter being less rigid and easier to correct.

In this, the common type, the sole looks inwards and backwards and in severe cases upwards inwards and backwards. The plantar flexion and adduction of the fore foot is shown by a crease across the sole and on the inner border of the foot at the level of the mid tarsal joint. The heel is inverted and drawn up owing to the associated equinus deformity. In severe cases the heel does not project behind this condition being always associated with a rigid and difficult foot to correct. Later, callosities develop on the outer border and on the dorsum of the foot from 'weight bearing' on these points.

In a club foot deformity the foot cannot be raised above a right angle at the ankle joint the sole cannot be turned so that it faces downwards and outwards and the inner border of the foot cannot be made straight. All these manipulations are



FIG. 55.—Severe club foot. Note the elevation of the heel and the crease across the sole at the mid tarsal joint.

possible in a normal foot. The deformity may be present in one or both feet and when of the primary congenital type it is sometimes associated with other congenital defects. In



FIGS 56 and 57.—Severe double club foot. Weight is borne on the dorsum of both feet.

single cases there is almost always an associated calcaneo valgus deformity present in the other foot needing treatment as urgently as in the club foot.

At birth the tarsus consists almost entirely of cartilage



FIG 58.—Callusity caused by weight bearing on severe club foot.



FIG 59.—Left club foot, right calcaneo valgus deformity.

There are only two small round centres of ossification—one for the calcaneus and the other for the talus (astragalus).

In a congenital club foot these tarsal cartilages are partially deformed in shape and in addition are mostly in abnormal

positions so that if a corrective force is brought to bear before ossification takes place the chances of obtaining a normal foot are very much better than if ossification is allowed to occur in the position of the deformity.

**Treatment.** Treatment should begin as soon as possible after birth. This is usually at about the age of ten days. Under the very best conditions and in a moderately easily correctable case the minimum time of treatment is about two years. Some cases—especially those which are associated with other primary congenital deformities, spina bifida, etc.—may be impossible to correct by manipulation alone even though treatment starts at the earliest age. This may be due either to powerful rigidity making it impossible to move the misplaced tarsal cartilages in relation to each other, or to the skin being unable to bear any pressure without breaking down. When the skin breaks down in this manner there is usually present some interference with the trophic nerve supply to the foot, such as may occur in an associated occult spina bifida deformity.



FIG. 60.—Primary congenital type of club foot associated with spina bifida.

In the earliest stage treatment consists of repeated manipulations to the foot of a special nature followed by strapping, so as to retain it in the maximum position of correction without interference with the circulation. These manipulations should be given about three times a week. It has never been found practicable to educate the parents to attempt these manipulations themselves. The adduction of the fore foot is the first deformity to correct and the equinus is the last.

As the child gets bigger, a shoe is fitted in order to obtain gradual correction. This shoe puts a gradual stretch upon the foot, evverting it and correcting the equinus deformity. It is worn day and night, and the amount of tension put on the foot

can be adjusted. Frequent manipulations are continued during the wearing of this shoe.

When the foot is fully corrected the position is maintained in a club foot shoe. This shoe has a sole piece with its fore foot abducted at the mid tarsal joint level, everted and placed above a right angle in relation to the leg.

Instead of using gradual correction shoes and night shoes plaster of Paris casts frequently changed give excellent results in skilled hands.

When walking commences the child should wear boots with this appliance fitted: an internal iron external malleolar strap, external toe raising spring and  $\frac{1}{4}$  inch rise on the outer side of the sole and heel. The appliance cannot be expected to correct any deformity which has been incompletely corrected by manipulation. It should however maintain a position of full correction when this has been obtained and while the evertors of the foot are receiving special treatment. This walking appliance must be worn during the day and the club foot shoe at night until cure results.

A cured deformity will show these characteristics: the foot will have a straight inner border and the child will be able actively to evert the sole and to raise the foot above a right angle with the leg. It will take at least twelve months' walking in the corrected position before cure can be obtained.

In the intractable club foot and in those which have been referred for treatment after walking has been allowed in the deformed position for some time open operations of various kinds are necessary. No open operation should be done however until the maximum correction by other means has been obtained. Up to puberty these operations are best confined to the soft structures of the foot and after such operations repeated manipulations or continuous gradual correction will be required.

If the foot is put up in plaster of Paris in the position of full correction obtained during an operation on the soft parts the skin will slough, the wound will not heal for a long time and a considerable increase in scarring will result. It is never possible by an operation on the soft structures to obtain full correction at once of the deformities.

If treatment is delayed until after puberty, a severe deformity results, and operations on the bony structures of the

feet must be considered. Following these operations the foot has considerable loss of mobility in its joints and will become more or less of a block foot according to the severity of the operation performed.

Very careful consideration is required before any operation is performed on the bones of these feet. Frequently more harm than good results and such harm is irremediable.

The most difficult cases of all are those which have previously undergone an operation on the bones and in which the deformities have recurred, or not been corrected. The results in such cases are deplorable.

The results of treatment in club foot deformity are on the whole, only fair. Taking all types and degrees of deformity together, about 50 per cent. can be expected to give a very good result both functionally and anatomically. It must be borne in mind that a good functional result does not necessarily require a perfect anatomical reposition and conversely, a good æsthetic result—especially if it follows bone operation—may prove very disappointing from a functional standpoint.

The treatment of the other types of congenital deformity of the feet follow roughly the same lines as in club foot. It is a universal rule in the treatment of these cases that the earlier treatment starts, the better will be the result.

Small babies cannot be admitted easily into orthopædic hospitals and it is only when a widespread orthopædic service is established through the country and babies can be brought for frequent treatment that the best results with the least expense will be obtained.

### The Knee Joint and Legs

Congenital abnormalities of the knee joint are rare. Occasionally congenital absence of the patella occurs or a patella may ossify from two centres. Usually the function is little if at all impaired, and the condition may safely be left alone.

Other congenital abnormalities such as congenital dislocation of the knee joint or congenital absence, or possibly a rudimentary form of one of the leg bones, are very rare conditions and will be readily recognised.



FIG. 61.—Double congenital absence of the patella

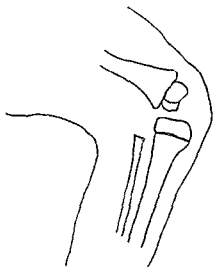


FIG. 62—x-Ray of knee joint. Same case as Fig. 61.



FIG. 63—Congenital deficiency of the left femur.



FIG. 64—Appliance fitted to compensate for shortening

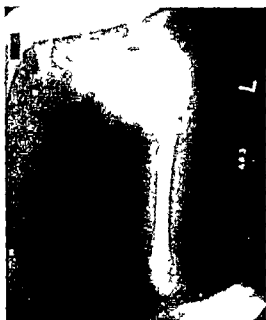


FIG 63—X Ray of congenital abnormality of the left femur (see Figs 63 and 64)

## THE HIP JOINTS

### Congenital Dislocation of the Hip

This condition is much more common in females than in males (about 11 to 1) and is more frequently unilateral than bilateral. It may be either a primary or secondary congenital deformity. In the former it may be associated with other congenital abnormalities; it will be very difficult to reduce and will be associated with considerable anatomical changes when examined by X rays.

In the secondary congenital type the dislocation has occurred in a hip which was developing normally and is due to some extraneous cause such as malposition in *utero*, deficient liquor amni, etc. The secondary changes which occur in this type are due to deficient development in the normal structures consequent upon their abnormal positions. It is associated with less anatomical change than the primary type and is more easy to reduce. The majority of cases are of this type.

**Pathology** The pathological changes which make reduction difficult are in most instances the direct result of late diagnosis and are aggravated by walking on the dislocated leg.

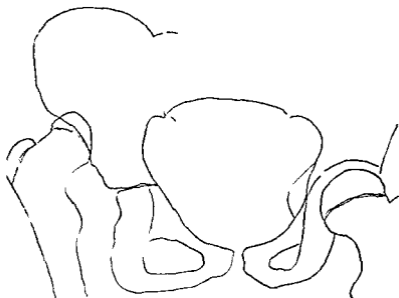


FIG. 66—Congenital dislocation of the hip. Note defective ossification of the head and deficient acetabular rim.

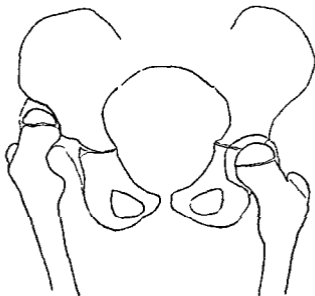


FIG. 67—Congenital dislocation of the hip. The dotted line represents the capsule.

(2) The head of the bone may be under developed and smaller than normal

(3) The neck of the femur may form almost no angle with the shaft & a condition of coxa valga may exist

(4) A deformity occurs in the neck of the femur in which the head of the bone is twisted forwards. This is termed ante version of the neck.

**Symptoms** Congenital dislocation of the hip is practically never diagnosed until the child starts to walk. Most cases are referred for treatment at the fourth or fifth year of life though many are not sent until they are eleven or even fourteen years of age.

(1) *Delayed walking*

(2) *True Shortening* Whenever real shortening is found in a limb measurements being taken between the anterior superior spine and inner malleolus of the ankle it is important to determine whether this shortening is situated above or below the great trochanter of the femur. This may often be difficult to discover.



FIG. 69 — Shoenemaker's lines. Congenital dislocation of the hip.

Of all the recognised tests in this connection *Shoenemaker's lines* are probably the easiest to apply and of the greatest help.

With the patient lying on his back marks are made over corresponding positions on the great trochanters of the femurs and on the anterior superior spines. With the aid of a tape lines are then drawn on each side starting at the great trochanters running

through the anterior superior spines and on to the mid line of the abdomen. Normally these lines will cross the mid line of the abdomen at or above the umbilicus. If there is shortening above the great trochanter on one side the line on that side will cross the mid line below the umbilicus. When shortening is present on both sides both lines will cross the mid line below the umbilicus.\*

\* A simple method of determining unilateral shortening is to lay the child on his back and flex the hips and knees when shortening on one side will be immediately apparent. This manoeuvre should be performed on every child soon after birth.

(3) *An Abnormal Gait* This is more easily noticed in a case of unilateral dislocation. When the child steps upon the dislocated leg the weight of the body causes the head of the femur to ride up on the ilium and the pelvis falls.

This abnormality can be best seen by the application of



FIG 70 — Trendelenburg test. When standing on the normal leg the opposite side of the pelvis rises.



FIG 71 — Trendelenburg test. When standing on the dislocated leg the opposite side of the pelvis falls. Same case as Fig. 69.

*Trendelenburg's sign* one of the most important clinical tests in the condition.

When the patient stands on a normal leg the pelvis on the opposite side either rises slightly or stays horizontal. When the patient stands on the dislocated leg the pelvis on the other side drops.

This test is only positive in a few other conditions, none of which will be present in a very young child. The dropping of the pelvis on the other side when standing on the dislocated leg is due to the fact that the head of the femur is not firmly fixed,

and the gluteal muscles are unable to act the fulcrum being unstable

If this test is found positive in a child who can abduct his legs outside the body line who has never had arthritis in his hip joint leading to bony ankylosis, and who has no infantile paralysis that child is suffering from congenital dislocation in his hip



FIG. 72.—Lordosis in bilateral congenital dislocation of the hip

When a double dislocation is present no matter on which leg the child stands the same result is obtained the pelvis on the other side dropping below the horizontal and thus giving rise to a marked waddling gait when the child walks

(4) *Telescopic Sign* If the pelvis is firmly fixed by an assistant and the leg grasped the femoral head can be pushed up and down on the outside of the ilium This sign is noticed more after the child has walked for some time—is the telescopic movement will increase with weight bearing It is almost absent in those cases which are due to a primary congenital defect as they are more fixed and in those who have developed a well marked false acetabulum in which the head of the femur is partly held

(5) *Lumbar Lordosis* In bilateral cases there is usually an increased lumbar lordosis as the weight has been transmitted more posteriorly and the pelvis tilts forwards There is also a widening of the perineum as the legs are placed further apart than normal

(6) *Position of the Head* The head of the femur can some times be felt on the dorsum ili—especially with the hip in the position of flexion and adduction—while there is an abnormal hollow in Scarpa's triangle

**Treatment** The earlier a hip is reduced the better will be the subsequent development and the more perfect the ultimate function

Up to the fourth year of age reduction is usually fairly

simple. It can be accomplished without the use of any force and the results are good. As the years go by it becomes increasingly difficult.

As a preliminary to reduction some surgeons consider mobilising exercises and manipulations to be of great value. These are intended to stretch the shortened muscles and ligaments round the joint and to free the capsule from any attachment it may have contracted with the ilium above the acetabulum.

Reduction of all dislocations is first attempted by manipulation. As in the treatment of all congenital deformities extreme gentleness is the secret of success. The more noise the actual reduction makes the more trauma has been produced. The hip if possible should slip in almost silently. The necessary stretching of the muscles before reduction is attempted should be very slow. The whole manipulation may easily take an hour or more.

It is difficult to say what is the greatest age when manipulative reduction can safely be attempted. Up to five or six years of age in expert hands it is safe to attempt reduction in bilateral and possibly up to nine years it is worth trying in the unilateral cases. Age in this connection is not the only factor to be considered. Usually the greater the telescopic movement the greater the chance of reduction as good range of movement shows that organic shortening of the vessels, nerves and muscles is not marked. If there is no telescopic movement and considerable real shortening reduction will be impossible and if force is used the circulation and nerves to the limb will be jeopardised.

If in a case where manipulative reduction is considered possible but where an attempt at correction has been followed by failure a constriction of the capsule is usually present and the case will require open reduction. Most authorities are against open reduction unless manipulative reduction is found impossible, for although stability will be given to the hip if the operation is successful, mobility will be restricted to some extent.

The operation for open reduction of a congenital hip should only be attempted in a hospital properly equipped for orthopaedic work. Many difficulties may be encountered during the operation which will call for all the skill and experience which the surgeon has at his command.

Whether reduction is accomplished by the closed or open method the whole treatment will occupy anything from eighteen months to two years. Up to six months or longer the position should be maintained by plaster of Paris casts which should be changed every three months. Subsequent development in the hip should be watched by repeated x ray photographs.

### CONGENITAL COXA VARA

The true congenital form of this condition is very rare though it sometimes runs in families as a primary congenital abnormality. It will be recognised by a limitation in abduction and if unilateral by a shortening of the leg which will be situated above the great trochanter of the femur.

Coxa vara differs from congenital dislocation of the hip in the following ways —

- (1) There is no telescopic movement
- (2) The head of the femur is in the correct position
- (3) Trendelenburg's sign is negative
- (4) x Ray diagrams will show the femoral head in the acetabulum and a diminution of the angle between the neck and shaft of the femur which in children should be about 160 degrees.

### SPINE

Congenital abnormalities of the spine occur but are seldom noticed until the child reaches the age of five or six years.



FIG. 3—Congenital abnormalities in the cervical vertebrae.

There may be a scoliosis or a side bending rotation deformity. One shoulder may be lower than the other or the neck may be abnormally short.

In x ray examination reveals that there is an abnormality in the development of one or more of the bodies of the vertebrae. There may be congenital absence of half a vertebra, wedging of some of the vertebral bodies or abnormal fusion of different parts of the spinal column (see Plate XVI).

These are primary congenital abnormalities and consequently, in these cases, a search should be made for other abnormalities, such as accessory ribs, spina bifida, etc. The prognosis for complete cure is bad, but the deformity curve which is usually marked can be prevented from getting worse by the application of accurately fitted jackets and corsets as the child grows. These must be renewed every three months.

## NECK

### Congenital Torticollis, or Wry-Neck

**Ætiology** Congenital torticollis is due either to a developmental abnormality of the cervical vertebræ, in which case it is almost impossible to correct, or, as is far more common, to organic shortening of one sternomastoid muscle.

*x* Ray examination will be required to reveal abnormalities in the vertebræ.

The cause of the second type is not known. It was formerly considered as being due to an injury to one sternomastoid muscle during a difficult birth. This is not now considered to be the case.

In those cases where there is a swelling of the sternomastoid muscle the swelling is probably due to an effusion of blood caused by injury to an already shortened muscle.

The longer the deformity is allowed to remain the greater will be the secondary changes. These constitute —

(1) *Asymmetry of the face* This develops fairly soon after walking, and is chiefly noticed by the fact that the line passing through the two eyes is not parallel with that passing through the mouth. These lines converge on the side of the shortened



FIG. 74.—Congenital torticollis. The head is bent to the side of the deformity and the chin rotated towards the opposite side. The eye and mouth line are not parallel.

muscle If the deformity is corrected fairly early in life this asymmetry tends to correct itself

(2) Organic shortening of the ligaments and other muscles on the side of the contracted sternomastoid

(3) Bone changes in the cervical vertebrae and secondary scoliosis of the dorsal vertebrae

**Symptoms** The head is pulled towards the side of the shorter muscle and the chin is rotated to the unaffected side If an attempt is made to correct the deformity the shortened sternomastoid muscle stands out as a cord

**Treatment** If the deformity is slight an attempt may be made to obtain correction by repeated manipulations and by the application of an apparatus to hold the head in an over-corrected position This is seldom successful

Operative correction consists in the division of both heads of the sternomastoid muscle with its shortened fascial covering and in the maintenance of an over-corrected position This position has to be maintained until the gap in the divided muscle has completely healed without shortening and this will take about six weeks It must be followed by exercises and manipulations for a further six months as the condition is liable to recur

The child cannot be regarded as cured until he can move his head actively into the position opposite the deformity and maintain it in that position This is one of lateral flexion towards the unaffected side with rotation of the chin towards the affected side

### **Congenital Elevation of the Scapula, or Sprengel's Deformity**

This deformity is a primary congenital defect It consists in —

(1) An elevation of one scapula up to 4 inches above that on the opposite side

(2) A shortening of the scapula in its vertical axis so that it is broader across than it is long

(3) Abnormalities in the development of the ribs and vertebrae

(4) The formation of connections which may be bony in type between the scapula and the vertebral column (see Plate XVII)

**Treatment** Unless the deformity is very marked it is wisest to leave it alone Operation only gives a fairly satisfactory

result as the deformity is very liable to recur, and no operative interference is justifiable unless marked improvement in function is expected.

## HANDS AND FINGERS

### Club Hands

The club hand consists in a rigid malposition of the hand in relation to the forearm. There are various types.

(1) *Radial Club Hand*. Here the hand is deviated to the radial side where the radius may or may not be completely or

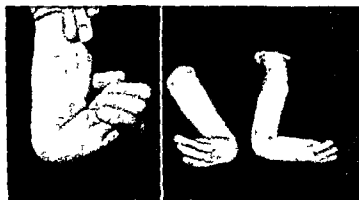


FIG. 75.—Ulna club hand

FIG. 76.—Palmar club hand

partially absent. This type of club hand is analogous to a rare and severe form of club foot in which there is a deficiency in the tibia

(2) *Ulna Club Hand*. To the ulna side.

(3) *Palmar Club Hand*. Forwards.

(4) *Dorsal Club Hand*. Backwards.

Combinations of these deformities occur, such as radio-palma club hand, etc.

**Treatment.** Treatment depends upon the degree of deformity and consists in gradual stretching and manipulations till correction is obtained. Sometimes in later life it is possible by bone-grafts to maintain correction in those cases where bone is deficient.

*Congenital Absence of the Hand* The forearm ends in a rounded stump and in some cases at the end of the stump are

situated small lumps which represent the rudimentary fingers possibly with minute nails. This stump although not



FIG. 7.—Congenital absence of the hand flexor surface



FIG. 8.—Congenital absence of the hand showing rudimentary digits

actually useless suffers from the great disadvantage of being unable to pick up or hold anything.



FIG. 9.—The prehensile forearm. Proximal approximates the digits so that articles can be held between them.

By bone grafts it is sometimes possible to construct a prehensile forearm. This attempt should not be made until the child is about twelve years of age and fully capable of giving active support and help in the development of its new limb.

*Cleft Hand or Lobster-claw Hand.* This consists of a defect in the centre of the hand usually

due to absence of some digits or even metacarpals with bifurcation



FIG. 80.—Lobster-claw deformity of the feet, dorsal view



FIG. 81.—Lobster-claw deformity, plantar view

of the hand. Operative interference with this type of hand is only indicated if the function can be improved.



FIG. 82—Congenital deficiency of the hand

*Polydactylism.* This consists in the existence of extra fingers or thumbs or parts of them. In most cases the extra digit is attached by skin only and should be removed, but in some



FIG. 83—Polydactylism in a child



FIG. 84 and 85—Polydactylism in an adult who has done heavy lifting work



cases it is a fully formed digit under complete control and, except for its unsightliness, does little harm. An x-ray photograph should be taken to show the bone condition present.

*Syndactylism, or Webbed Fingers.* The fusion between the fingers may be formed by skin or by skin and bone. Most webbing occurs on the ulna side of the hand.

*Treatment.* By the formation of flaps, when the webbing is due to skin, the fingers may be separated. In some cases skin grafting is necessary.

## SECTION VIII

### CHAPTER XVIII

L. B. SOMERVILLE LARGE

#### THE EYE DURING THE FIRST TWELVE MONTHS OF LIFE

(Percentage of Blindness—Defective Vision in Early Life—Methods of Examination—Ophthalmia Neonatorum—Ocular Trauma at Birth—Embryology—Aberrations of Development—The Eye at Birth and Later Development—General Conditions Occurring during the First Twelve Months of Life)

#### GENERAL

##### Percentage

As statistics relating to the blind population are based solely on those who are in receipt of the blind pension or blind relief and do not take into account the self supporting blind they are inaccurate both as regards incidence and causation of blindness. It is estimated however that 21.4 per cent of those certified as blind were blind before reaching the age of five and further more that two thirds of these were blind before they were a year old. Knowledge of ocular disease and defects occurring during this period is therefore of great importance.

##### Defective Vision in Early Life

In the early months of life it is difficult to determine the presence of defective vision. The baby with bad sight will not grasp or pick up toys, does not recognise those attending it and is seldom seen to gaze fixedly at any object. Later if short sight be present objects will be held close to the eye or laid on the ground and the face brought close to them. Children with bad sight are frequently seen when facing the light to move the outspread fingers from side to side across the face. This manoeuvre although indicating a low degree of visual acuity proves that the child is not totally blind as it produces a flicker effect on the retina showing that the presence of light is appreciated.

Blind or partially sighted children are backward and timid requiring very different methods of upbringing from those

normally sighted. As these methods are now well understood and as special organisations exist which apply their expert assistance should be sought immediately bad sight is recognised.

### METHODS OF EXAMINATION

Ocular examinations of babies and of children call for different technique.

A baby is examined lying on a table with a good light shining into the eyes. The examiner stands behind the head which an assistant, facing him, steadies between his hands. Both the examiner's hands are thus free to manipulate the lids or carry out treatment.

With children the assistant and examiner sit facing one another. The child's head is held between the examiner's knees; the assistant's lap supports the body while the arms are held firmly by the side. Here again the examiner's hands are free with the head held in perfect control.

In bottle-fed children advantage may be taken of feeding time, when an examination of the ocular fundi carried out under atropine mydriasis is often satisfactory although for a complete examination to be made a general anaesthetic is necessary.

### OPHTHALMIA NEONATORUM

By ophthalmia neonatorum we mean an inflammation of the conjunctiva of the new-born which in rare instances is present at birth but much more commonly occurs after it.

#### Incidence

The following figures show the importance of ophthalmia neonatorum in its relation to the blind population. This condition is found to be present in from 20 to 30 per cent of children attending schools for the blind and in 2 to 3 per cent of the adult blind. It is stated to occur in some 8 per cent of all births (Cardell).

It is notable that the incidence of ophthalmia neonatorum has not decreased in London in the last fifteen years (Mayou).

#### Causative Organism

In all cases of ophthalmia neonatorum a swab should be taken immediately and a direct examination made from a smear.

preparation It is of the utmost importance to remember that although the condition is frequently caused by the gonococcus this is by no means invariably so At one time it was considered that 60-65 per cent of all cases were gonorrhœal but the examination of 1 126 swabs carried out recently at the Royal London Ophthalmic Hospital showed the staphylococcus to be the commonest causative organism (Browning) The pneumococcus *Bacillus xerosis* and the bacillus of Koch Weeks may also cause ophthalmia neonatorum while a mixed infection is common The most severe conjunctivitis of the new born follows a streptococcal infection In gonorrhœal ophthalmias the diagnosis is readily made as the gonococcus is found in great numbers in the smear

### Defence Mechanism of the Eye at Birth

At birth the eye has less resistance to infection than in later life

In the new born the epithelium of the conjunctiva and cornea is thin and composed of fewer layers of cells thus presenting a weaker barrier to invading organisms The lymphoid tissue also which is normally present in the conjunctiva is absent at birth, and is not fully developed until the fourth week of life Lastly, the infantile eye is deprived of the important defensive mechanism of tears which have a valuable flushing and lysozyme action as the lacrimal secretion does not appear until after the first few weeks of life

### How Infection Occurs

Immediately after birth the lids are firmly closed and usually in accurate apposition The outside of the lids is covered with a film of greasy material and the margins with Meibomian secretion Ophthalmia results most commonly from contagion occurring during birth the infecting organism invading the lids during the second stage of labour Subsequently when the eyes are opened the conjunctivæ become infected Sometimes however the secretion is washed into the eyes with the first bath Should labour be prolonged infection is more likely to occur

Infection that is brought about during birth commonly manifests itself on the second or third day, but may be delayed

to the fifth A conjunctivitis that comes on after this time follows some extraneous infection, due in most cases to lack of cleanliness

### Description of the Condition

*Ophthalmia neonatorum* presents the same clinical features, whatever may be the infecting organism. The inflammation varies both in duration and severity with the virulence of the organism, corneal involvement being much more likely to occur from infections by the gonococcus and streptococcus. In the majority of cases both eyes are infected simultaneously.

The earliest sign of commencing ophthalmia is a redness about the inner angle of the eye. This is followed by an oedematous swelling of the lids and the commencement of a watery secretion resembling tears (again we must remember that at this early period of life no tears are formed) which coagulates on the lid margins, sticking them together.

Next the lids become acutely inflamed and greatly swollen, with the skin stretched tightly over them and the margins glued together by a yellow discharge. On opening them, thin creamy pus flows out on to the cheeks. Sometimes this gluing of the lids is so complete that the pus is held back under considerable tension, and is squirted out when the lids are opened, to the great danger of the examiner's eyes. Hence it is essential that protective goggles be worn at every examination and treatment during this stage of the condition. When the pus is bathed away the conjunctiva itself may be examined. It will be found to be greatly inflamed, swollen and roughened. That of the globe itself may be so oedematous as to overlap the margin of the cornea, producing the serious condition of chemosis. In severe cases a membrane, that can be peeled off, forms on the conjunctiva. The pre auricular gland is enlarged.

This acute condition gradually passes into the stage where the lid oedema goes down, and the normal skin folds make their appearance again, the discharge becomes diminished and the conjunctival inflammation steadily decreases. If the discharge suddenly becomes less or ceases, a serious loss of resistance is indicated, with danger to the child's life.

The great danger of ophthalmia neonatorum is corneal ulceration, which is stated to occur in 27 per cent of cases. The healed ulcer leaves a corneal scar which interferes with

vision to an extent depending on the size and position of the area involved. Should the ulcer perforate as frequently occurs the sight will be greatly and permanently damaged.

The general symptoms of ophthalmia neonatorum are a rise of temperature and a retardation of the normal increase in weight. Thus the mortality of premature infants with this infection is very high. In rare cases an acute arthritis or a subacute synovitis have developed towards the end of the second or third week. It is not common to find congenital syphilis in association with ophthalmia neonatorum. Its presence adds considerably to the danger of corneal involvement and raises the mortality rate. Infection spreading from the naso-lacrimal duct to the nose gives rise to the rhinitis that is commonly present.

### Prophylactic Treatment

The prophylactic treatment of ophthalmia neonatorum consists in the cleansing of the lids at birth followed by the instillation of germicidal drops into the eyes.

Ophthalmia neonatorum is a notifiable disease and as it is highly contagious its notification should never be delayed.

Midwives receive careful instructions regarding its prophylaxis and diagnosis and in some districts the putting of germicidal drops into all baby's eyes at birth is compulsory. This measure has everything to recommend it and there can be little doubt that its general adoption would lower the incidence of the infection. All cases of ocular inflammation and discharge occurring within the first two weeks of life however slight must be reported to a doctor immediately.

The most important prophylactic measure is the treatment of the eyes immediately after birth. As soon as the child is born and before the eyes are opened each eye should be wiped with separate pieces of dry sterilised cotton wool until all the secretion present on the lid margins is removed. If the wool be dry as well as removing this secretion it also removes the grease that is normally found on the lids thus allowing them to be more easily manipulated during the subsequent insertion of drops. Moist swabs have the danger of washing the secretion into the eyes. *This procedure should never be omitted and reliance placed solely on the use of drops.*

Next drops are put into both eyes the baby's head being controlled in the manner previously described (see p. 399).

The drop must be seen to enter the conjunctival sac. One drop only is necessary, more than this runs on to the cheeks and may cause excoriation.

Various antiseptics have been suggested but there can be little doubt that the surest is a 1 per cent solution of silver nitrate. This drug is more strongly germicidal than any other that can be used in the eye with safety. The solution should be kept in a coloured bottle and renewed at least once a month, as its germicidal action is lessened by decomposition on standing.

The objection to silver nitrate is the local ocular reaction that it sometimes causes. This reaction may be mistaken for an attack of ophthalmia neonatorum or conversely be considered as due to the drug when it is actually a manifestation of the disease. The two are readily distinguished. The reaction from silver nitrate commences within a few hours after birth and steadily subsides to disappear altogether in forty-eight hours while that from ophthalmia neonatorum does not commence until the second day and steadily increases. Ocular damage from the use of 1 per cent silver nitrate is unknown. It has been in use for many years as a prophylactic routine in the Rotunda Hospital here also a second installation is given some two hours after birth if the mother is found to have any abnormal vaginal discharge. Inflammatory reaction from its use occurs in this hospital in less than one case for every thousand babies treated.

As it is not possible to exclude the risk of vaginal infection too much emphasis cannot be laid on the importance of the routine employment of these simple and harmless measures.

Pregnant women should be questioned regarding leucorrhœa and if it is present to any degree a swab must be taken and the necessary treatment instituted. After birth emphasis should be laid on the necessity for scrupulous cleanliness of all clothing that comes in contact with the baby's head, for a conjunctivitis may occur at any time. A baby who escapes ophthalmia at birth although the mother has a gonorrhœal discharge, may well contract it at a later date unless much care is exercised.

It is of great importance to keep a close watch during the first days of life for any signs of ophthalmia, as a few hours' delay in treatment may well mean the loss of an eye. It

must be remembered that statistics of blindness are based on the loss of sight in both eyes the percentage of damage to or loss of one eye only is considerably higher

As these cases are of the utmost gravity it is strongly advisable to seek expert ophthalmic advice whenever possible

To sum up the prophylactic treatment of ophthalmia neonatorum consists of careful cleansing of the lids at birth followed by the installation of 1 per cent silver nitrate solution

**General Treatment** As will be seen from the following the local treatment of ophthalmia neonatorum must be carried out thoroughly and frequently or serious loss of sight will result It will then be readily understood that the condition is most unsuitable for management at home and all cases should have inpatient institutional treatment It is advisable for nursing mothers to be admitted with their children and when necessary to receive treatment for their vaginal discharge

Breast feeding should be insisted upon in all cases unless strongly contra indicated It has been shown beyond doubt that with breast feeding the inflammation clears up much more rapidly than when artificial feeding is instituted and also that corneal complications are less common

Fresh air and good hygienic conditions play a valuable part in the treatment of the condition

Vaccine therapy appears to have no value in the treatment of ophthalmia neonatorum

**Local Treatment** It cannot be too much stressed that the treatment of ophthalmia neonatorum must be commenced immediately the diagnosis is made as delay of even a few hours may seriously affect the result

When the diagnosis is made a swab should be taken at once This should be carried out before any treatment is commenced for once any germicidal treatment has been used some hours must elapse before it can be done with any hope of obtaining a culture If no organism is obtained from the first swab it should be repeated The virulence of the infecting organism is now established

Thorough examination is essential and is carried out in the manner described above (see p 399) The pus is swabbed away and the lids gently opened Should they be much swollen protective goggles are to be worn by both attendants to guard against a squirt of pus entering their own eyes Lid retractors

(Fig 86) are now inserted and the cornea carefully examined. It is impossible in the presence of lid œdema to examine the cornea completely without retractors. If the cornea is not uniformly bright a drop of fluorescein (2 per cent) followed by a few drops of boric lotion is instilled into the eye. As a slight degree of dullness of the cornea is not easily recognised it is wise to carry out this procedure as a routine. Should any ulceration however small be present the area involved will now be stained bright green.

Local treatment consists of irrigation, the use of drops and the application of silver nitrate.

Frequent and thorough irrigation of the eye is the secret of successful treatment. It is carried out in the position described above.) An undine is used (Fig 87). If

the right eye is to be treated the head is held slightly turned with this eye down and a receiver held against the cheek to catch the irrigating fluid. The lower lid is retracted by the

assistant's left forefinger and the upper lid by the right forefinger of the nurse administering the treatment whose left hand is thus free to manipulate the undine. A corresponding position is used for the left eye. Sufficient fluid is used to

wash out the conjunctival sac completely. prolonged irrigation has no value and may damage the corneal epithelium. Irrigation is carried out every hour in severe cases which have much discharge and only two or three hourly when the inflammation is less acute. As the discharge lessens the treatment is carried out less frequently for the condition is both aggravated and prolonged by over-treatment.

Many irrigating fluids have been recommended. the success of this treatment, however, depends more on the method of its application than on the drug employed. It is well to commence with a mild lotion composed of boric acid and sodium bicarbonate (3 per cent) for the first twenty four hours and then continue for the rest of the treatment with a solution of oxy cyanide of mercury (1 10 000). The solutions are used warm



Fig 86 — Lid retractor

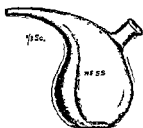


Fig 87 — Undine

After every irrigation the drops are inserted. These should either be one of the silver preparations (argyrol, 25 per cent, and protargol 15 per cent) mercurochrome (2 per cent), or acriflavine (1 in 1,500). It is well after using one of these to follow it with an oily drop, either paroline or castor oil as this keeps the lids from sticking together and helps the discharge to find its way out between them. Acriflavine made up in paroline is a most satisfactory solution. The silver preparations should not be continued for more than six weeks, as they may give rise to the condition of *argyrosis of the conjunctiva*. Silver nitrate in the strength of 10 gr. to the ounce should be painted on to the conjunctiva of the fornices and of the everted lids by means of a piece of wool mounted on a matchstick. It has little value when used as a drop. It must not be applied in the acute stages as it aggravates the condition. Its application is commenced when the lid œdema is going down and the skin folds making their appearance. From then until the stage when the discharge has greatly diminished it should be applied daily.

Between irrigations the lids must be swabbed clear of any secretion that may form on them. When the lid œdema is very intense the palpebral aperture can be enlarged with a single snip of a strong scissors through the outer canthus (canthotomy). This little operation is of great value and should not be delayed if the degree of œdema is such that the pus is held back under pressure. It allows the irrigation of the conjunctival sac to be very much more easily and thoroughly carried out. The resultant scar is not visible.

If corneal ulceration is present a drop of atropine (1 per cent) twice daily should be added to the treatment. The symptoms of atropine poisoning must always be kept in mind in these cases, i.e., dryness of the mouth and throat, a local skin condition resembling impetigo and rarely, a mild delirium. If any of these occur its use must be immediately discontinued. A spreading ulcer is treated by having its base and sides touched with a pointed match dipped in pure carbolic acid.

It is well to keep a piece of lint between the head and the pillow, it should be replaced as often as it becomes soiled with discharge from the eyes.

When one eye only is affected every precaution must be taken to avoid infection spreading to the other. It is kept covered by a close fitting pad of cotton wool held in position by

strips of adhesive plaster, this is only removed twice a day, when the eye is inspected and a drop of argyrol (15 per cent) is instilled. The baby should be kept continuously on the side of the discharging eye. All swabbing must be done from within outwards, and during irrigation the solution must not be allowed to overflow into the healthy eye.

To sum up the treatment of ophthalmia neonatorum mother and child should be admitted to hospital. the unaffected eye should be kept covered, all discharge should be frequently removed, and gentle irrigation carried out every few hours, followed by the instillation of drops.

### When to Discharge from Hospital

Babies with ophthalmia neonatorum require on the average a stay of four weeks as in patients and it is well not to send them out until the discharge has stopped for seven days. Recurrences are common, for the gonococcus can often be recovered from the conjunctiva up to four weeks after the discharge has ceased.

After treatment Cases of ophthalmia neonatorum should be seen at regular intervals after they leave hospital. In eyes with central corneal opacity iridectomy may be performed at about the sixth month to improve vision and prevent nystagmus. Later in life corneal transplantation holds out a possibility of visual improvement. It must be remembered that even dense opacities may clear up to a remarkable degree.

If the globe is quite destroyed and becoming shrunken and unsightly it should be removed.

### OCULAR TRAUMA AT BIRTH

Ocular damage may occur during birth the most serious damage following the incorrect application of the obstetrical forceps.

#### Injuries during Normal Labour

Oedema of the lids is common being sometimes very extensive and ecchymosis can also occur. The conjunctiva may show sub-conjunctival hæmorrhages and inflammatory oedema.

Retinal hæmorrhages are found as a fairly frequent accompaniment of normal labour. They are present throughout the

fundus and are situated in the inner layers of the retina. They are absorbed without producing permanent damage.

A retrobulbar hæmorrhage resulting from a fracture of the orbit and due to forcible uterine contractions may occur at birth.

Ocular damage has been known to result from palpation of the orbit in mistake for the foetal anus when attempting to diagnose the presentation.

### Forceps Injuries

Excoriations, unilateral exophthalmos and paralysis of the lids muscles in association with a fracture of the orbit have been produced by the blade of an incorrectly applied forceps.

The cornea may show either a diffuse opacity as a common temporary condition following pressure œdema or a deep opacity caused by the rupture of the posterior elastic lamina (Descemet's membrane) from direct pressure of the forceps blade. The latter injury results in a permanent loss of sight.

Hæmorrhages may occur into the anterior chamber (hyphæma) from damage to the iris or ciliary body. The lens may suffer a traumatic cataract.

The retina may show either hæmorrhage or œdema. The latter disappears without permanent damage. The former can also be absorbed but if large sometimes produces a retinal detachment with consequent serious loss of visual function.

Optic atrophy may result from an orbital fracture extending into the optic foramen.

Paralytic squint and ptosis may be produced from injury to the sixth and third nerves respectively. The former is the more common.

### EMBRYOLOGY

At the 3.2 mm stage of the developing embryo the optic pits appear as depressions in the interior of the neural ectoderm forming the lateral walls of the fore brain. These deepen to form the primary optic vesicles (Fig. 88). Invagination of these commences below and to the outer side by the formation of the foetal fissure which gradually deepens to produce the secondary optic vesicles. It is during this period of development that colobomata of the iris, lens and choroid occur through the

failure of the foetal fissure to close completely. A thickening occurs, when this invagination commences in the surface mesoderm covering the extremity of the primary optic vesicle. This is the lens plate which being invaginated in its turn forms the lens vesicle (Fig 88). When invagination is complete it becomes cut off from the surface mesoderm and develops into the lens.

Later, differentiation occurs in the mesoderm surrounding

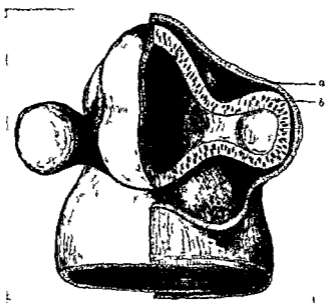


FIG 88.—Model of the fore brain and optic vesicles of a 4 mm human embryo seen from in front. The optic vesicle on the left of the embryo is represented in section. (a) Surface mesoderm (b) Neural ectoderm.

(Mann. Development of the Human Eye. By courtesy of the *British Journal of Ophthalmology*.)

the secondary optic vesicle to form the coats of the eye ball (i.e., the cornea and sclera.)

The neural ectoderm of the secondary optic vesicle gives rise to the retina, optic nerve, sphincter and dilator pupillæ muscles. These last are the only muscles in the body to be developed from this embryonic tissue, from which the central nervous system is also developed.

Thus it is seen that the lens is developed from the same part of the embryo that gives rise to the hair, skin and nails, and, like them, continues to grow throughout life.

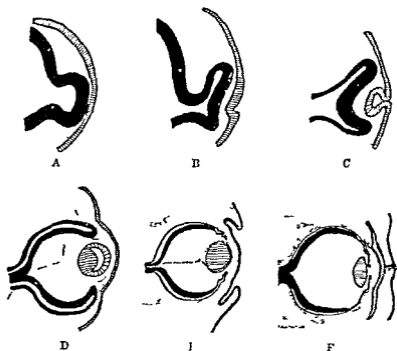


FIG. 89.—The normal development of the human eye. Neural ectodermal shown in black. Surface ectoderm is lined. Mesoderm is dotted.

(A) Stage of primary optic vesicle present as an outgrowth of the fore brain and in contact with the surface ectoderm.

(B) Stage of commencing invagination of the primary optic vesicle and appearance of the lens pit in the surface ectoderm.

(C) Stage of formation of the secondary optic vesicle and of the lens vesicle.

(D) Deepening of the secondary optic vesicle and separation of the lens vesicle from the surface.

(E) Commencing forward growth of the margin of the secondary optic vesicle to form the ectodermal part of the iris (the sphincter and dilator pupillae muscles and pigment epithelium).

(F) Eye complete.

(Duke Elder. Recent Advances in Ophthalmology. 1935.)

### ABERRATIONS OF DEVELOPMENT

A hereditary factor is found in a great number of developmental aberrations of the eye. Some ocular defects, however, although hereditary, may not develop until after the first year of life. It is stated that approximately 7 per cent of certifiable blindness is due to hereditary affections.

### Ptosis

This may be complete, through entire absence of the levator palpebræ superioris muscle. In these cases slight elevation of the lids can be produced by contraction of the occipito-frontalis. It is, however, more commonly partial, and is usually bilateral. Affected individuals are seen to adopt the characteristic position with the head thrown back so as to allow them to see below their half closed lids. Many operations have been devised for the successful relief of this condition. It is markedly hereditary.

### Epicanthus

Epicanthus, which is more commonly bilateral, is a condition in which a semi lunar fold of skin overhangs the inner angle of the eye, it is continuous with the skin of the nose. Children in whom it is well marked appear to have a convergent squint. As the nasal bones develop this fold of skin becomes lifted up and the condition may altogether disappear. It is seen both in the "mongolian facies," and in the necrosis of the nasal bones that is associated with congenital lues.

### Blue Sclerotics

Heredity plays an important part in this condition where on account of abnormal translucency of the sclera the ciliary body and choroid show through, giving the globe a blue appearance. It is associated with hypofunction of the parathyroid. Affected individuals may also suffer from brittleness of bone (*fragilitas ossium*) and otosclerosis.

### Buphthalmia

Buphthalmus, or infantile glaucoma, is caused by the imperfect development of the angle of the anterior chamber. The disease may be present at birth or may arise during the first year of life. On account of this defect the aqueous humour cannot escape from the eye, and the resultant rise in intra-ocular tension causes the sclera and cornea, which at that period are expansile, to stretch so that the whole eye is greatly enlarged. The globe protrudes through the lids, vision is very seriously reduced, and, if the condition is not arrested either spontaneously or by operation, complete blindness results. It is therefore essential that all cases be examined as

early as possible with a view to surgical interference. The defect is very markedly hereditary.

### Albinism

The albino shows marked deficiency of pigment in the uveal tract. The iris is thus rendered permeable to light and the red reflex of the fundus can be seen through it giving the eye a pink appearance. Macular fixation is never obtained so that nystagmus is invariably present and the vision very low. As there is no protective pigmentation photophobia is intense.

### Congenital Cataract

Congenital cataract shows many varieties classified as to the region of the lens involved. The most frequent is lamella cataract where the lens nucleus is opaque and the surrounding cortex clear. As convulsions may occur with this defect and also as the permanent teeth may show poor development with hypoplasia and horizontal ridging the condition is generally regarded on purely theoretical grounds as related to irregular calcium metabolism associated with parathyroid deficiency.

Lamellar cataracts in some cases do not appear until after birth. The ætiology of these is obscure but is considered by some authorities also to be associated with dysfunction of the parathyroid glands and irregular calcium metabolism. In this connection it is interesting to note that the parathyroid glands are not functioning in the human at birth and that therefore the child must obtain its parathyroid hormone through the mother's milk. In the calf on the other hand the parathyroids are secreting at birth and hence cow's milk lacks this product. Therefore it is supposed that babies fed on cow's milk are more prone to lamellar cataract than breast-fed babies.

These infantile cataracts both congenital and those developing after birth may be hereditary.

The degree of impairment of sight resulting from congenital cataract depends on the site and density of the opacity. If vision is much reduced the operation of needling of the lens is performed. If however the sight be 6/18 or better operative interference should not be undertaken. Should there be no red reflex through the lens or only a very peripheral one it is

well to operate before the sixth month of life with a view to acquiring fixation and preventing the development of nystagmus

The operation consists in dividing the lens capsule and thereby allowing the aqueous humour to come in contact with the lens fibres, causing them to swell up and ultimately be absorbed. Cataract glasses have then to be worn throughout life

### THE EYE AT BIRTH AND LATER DEVELOPMENT

At the time of birth the eye and brain are considerably nearer to their mature state than the rest of the body. From birth to maturity the body as a whole has to increase in volume twenty one times, while the eye and brain have but to grow three and a half times for their full development to be reached. The most rapid period of ocular growth is during the first year of life. The anterior part of the eye develops more rapidly than the posterior, the cornea reaching its normal size by the end of the second year.

In European races the iris stroma is unpigmented at birth and the eye is blue. This colour is due to the light which on entering the eye, becomes reflected through the unpigmented iris stroma by the underlying pigment epithelium (*i.e.*, the phenomenon of interference). Later, should pigment develop, the iris changes in colour through varying shades to dark brown, depending on the quantity of pigment present. In the coloured races this pigment is present at birth and the eye is consequently brown.

At birth the pupil normally contracts in response to light, and momentary unocular fixation can usually be elicited. The duration of this fixation gradually increases until, at the end of five or six weeks, momentary binocular fixation may also be obtained. This fixation is very loose, and a fleeting squint can frequently be observed. By the sixth month binocular vision should be established.

It will be observed that babies have an unblinking fixed gaze. The normal blinking reflex, which occurs from two to three times a minute in the adult, is not developed until about the sixth month, and the light blinking reflex not until the ninth.

The antero-posterior diameter of the globe is less at birth

than in the fully developed eye. Hence the eye at birth is flattened and the refraction hypermetropic.

Tears are not secreted for from fourteen to twenty-one days after birth, lubrication of the eye being carried out by the accessory lacrimal glands of the conjunctiva.

### GENERAL OCULAR CONDITIONS OCCURRING DURING THE FIRST TWELVE MONTHS

For the first year of life ocular hygiene consists in the protection of the eyes from light infection and mechanical trauma. Owing to the late development of the light blinking reflex (ninth month) the eyes should be carefully protected from exposure to direct light. Prams should be hooded and not left facing the light and later shady hats worn. The wearing of dark glasses cannot be recommended in Western climates as when they are discarded the eyes are supersensitive to light. As the conjunctival resistance is low care has to be taken to avoid allowing contact with soiled clothing and linen. It seems superfluous to insist that no child should be allowed access to anything that might injure the globe but many young eyes are still lost annually by means of such domestic instruments as the scissors and table fork.

#### Strabismus (Squint)

We have seen that the power of binocular fixation should normally be acquired by the end of the first six months of life and that up to then one eye may be seen to turn independently of the other. Should a squint be observed after this time it must be regarded as pathological and whether periodic or continuous in character should be treated without delay. Children who grow out of a squint do so at the loss of much sight in the squinting eye.

There is no reason why refraction may not be carried out under a mydriatic and glasses worn during the first year of life but it is usually sufficient merely to paralyse accommodation in the non squinting eye so as to enforce fixation in the squinter. With this treatment careful observation is essential so as not to change the squint to the other eye. Orthoptic treatment whereby the eyes are trained to require binocular vision cannot usually be started until the end of the second year.

The condition shows a strong hereditary tendency

Apart from this, where the eye itself is normal it must be remembered that some abnormal conditions of the eye occurring at this period of life may produce a squint. Thus an eye with a glioma or cataract may deviate or a paralytic squint result from a birth injury to the external rectus muscle. It is essential therefore that every squinting eye be thoroughly examined

### Measles

Eye complications are of frequent occurrence during an attack of measles. The commonest is conjunctivitis which if serious may proceed to corneal ulceration. The local treatment consists in the instillation of a drop of argyrol (15 per cent) to both eyes twice daily with the application of ung bor to the lid margins to prevent excoriation. Keeping the room darkened has no beneficial effect on the eye but if there is much photophobia direct light may be excluded from the face by a screen beside the bed. The presence of light and fresh air in the room do not predispose the eyes to inflammation

### Vaccinia

Following vaccination the lids sometimes become very œdematous small ulcers often developing. Secondly to this inflammation of the lids the cornea may become ulcerated and although the lids themselves readily heal the corneal ulceration may be exceedingly resistant to treatment

### Glioma

Ocular glioma (neuroblastoma) is a primary malignant neoplasm of the retina. It is found only in children usually before the fifth year of life and in some 50 per cent of cases before the end of the second year. The condition has been found to be present so soon after birth that it must have developed *in utero*

Glioma spreads directly to the brain along the optic nerve. Metastases are rare. Death does not usually occur for several years

The first change commonly noted is in the appearance of the pupil, which instead of being black is grey or white with a

golden yellow reflex. There is no inflammation. It can be mistaken for either a pseudo glioma (where there is a yellow organised exudate in the vitreous following inflammation) or for congenital cataract. It is frequently bilateral and may show a high familial tendency, several children in the same family being affected. As we have seen, an eye with a glioma may squint and this is often the first indication of the condition.

The treatment consists of removal of the eye immediately the diagnosis is made.

The remaining eye will have to be kept under constant observation until the child is at least five years old, and if a glioma occurs in it this eye must also be removed. Should however this be refused and the growth be small, it may sometimes be completely destroyed by the local application of radon seeds and useful vision be retained.

### Lacrimal Obstruction

Sometimes at birth the naso lacrimal duct is not patent, being blocked by a plug of cells at its lower extremity. Thus the tears cannot drain normally into the nose, and the eye is constantly watering.

A muco purulent discharge may be present and on pressure over the lacrimal sac muco pus can often be expressed through the lower punctum into the conjunctival sac. This differentiates the condition from ophthalmia neonatorum.

Treatment consists of pressure applied many times during the day by the finger over the sac against the side of the nose. This by compressing the sac tends to force the plug of cells out. If it is not effectual the duct will have to be probed.

### Congenital Nystagmus

The so-called congenital nystagmus is not usually present at birth, but develops within the first few months of life. It is a manifestation of some ocular abnormalities such as cataract, albinism or disease of the retina and choroid. Its treatment is that of the causal condition.

## SECTION IX

### CHAPTER XXIV

T G WILSON

#### CONGENITAL ABNORMALITIES OF THE EAR NOSE AND THROAT

(Deaf Mutism—Ætiology—Hereditary Acquired Pathology Diagnosis Treatment—Congenital Malformations—Macrotia Microtia—Congenital Stenosis Occlusion of the Anterior Nares Occlusion of the Posterior Nares—Dermoid or Hairy Polypus of the Nasopharynx—Congenital Laryngeal Stenosis—Congenital Stenosis of the Œsophagus—Congenital Imperforation of the Œsophagus)

##### Deaf Mutism

(1) **Hereditary** Congenital defects of the external middle and internal ears vary greatly in degree. Many important structures in the cochlea may be absent or rudimentary. Most cases tend strongly to be transmitted.

(2) **Acquired** Syphilis is the most important cause of acquired deaf mutism but as Taylor has pointed out ante natal medication *e.g.* with quinine may be a more important factor than is supposed. Measles and scarlatina are also common causes. Mumps may cause severe incurable deafness without supuration.

In acquired deaf mutism the static and acoustic labyrinths are usually both destroyed in distinction to the congenital cases in which the defect is mainly in the cochlea.

The diagnosis cannot be made with certainty before the sixth month even in normal children. In infants testing is carried out if possible without the child being aware of the examiner's presence. The latter should stand behind a curtain and on the child being brought into the room make a noise with a whistle or rattle. If the child makes a movement in response to the noise or turns his eyes in the direction of the curtain he has probably heard something. Caloric or turning tests will ascertain the condition of the semi-circular canals. Older children can of course be subjected to more complicated audiometric and other tests. If a limited amount of hearing is

found auditory education and speech training should be begun at once

### **Congenital Malformations of the External Ear, or Auricle**

These are many and they vary greatly both in type and importance. The auricle may depart considerably from the normal standard of size without attracting attention so long as both ears are similar. If on the other hand one ear differs noticeably from the other in size or projects more from the side of the head a real deformity calling for correction exists.

#### **Macrotia**

In this condition the auricle as a whole is enlarged the cartilage is unusually broad and the lobule may be very large and presenting forwards. Darwin's point may be unusually marked and project upwards and backwards from a large flat ear giving a satyr like appearance. Again the ear may project abnormally from the side of the head.

Supernumerary appendages are seen fairly often usually in the form of small cartilaginous nodules covered with normal skin in fairly close proximity to the auricle. They usually appear immediately in front of the tragus or below and beneath the lobule.

In these conditions the hearing is usually perfect and correction is undertaken wholly for cosmetic reasons. Plastic operations give very successful results but should not be undertaken before the seventh or eighth year by which time the external ear will be developed to approximately its adult form. It is important that an ugly deformity should be removed as it usually leads to the child being teased at school and may actually interfere with his future career.

#### **Microtia**

This is a much more serious condition. The term denotes abnormal smallness of the auricle and the condition is often associated with atresia or stenosis of the external auditory meatus. The external ear may be represented by a shrivelled shapeless bar or bars of cartilage with a short blind fossa replacing the external meatus. It will be obvious at a glance

that no plastic procedure could produce a normal looking auricle in these cases. The condition however, is often bilateral and if so the more important question arises as to whether any operation can be undertaken for the improvement of the hearing thereby saving the patient from lapsing into the condition of deaf mutism. As a rule the internal ear in these cases is developed normally and one might conclude from this that plastic operations directed towards the formation of an external meatus should be successful in producing greatly improved hearing. In practice however a few brilliant results have served only to emphasise the fact that operation is usually a failure. This is because the middle ear cavity is malformed and rudimentary the ossicles sometimes being enmeshed in dense connective tissue. In the presence of bilateral microtia with a moderate amount of hearing operation should be carried out on one ear at a time. If the formation of a meatus in the normal position is not possible permanent mastoid antrostomy may be performed. Hearing tests are of course extremely unreliable in children and the surgeon may have to operate without knowing how much useful hearing is present. If however, there appears to be the least possibility of effecting an improvement operation should be advised unhesitatingly.

## CONGENITAL NASAL STENOSIS

### *Congenital Occlusion of the Anterior Nares*

This is an uncommon condition in which a membrane is found in the nasal vestibule partially or completely blocking one or both nostrils at the junction of the skin and nasal mucous membrane. The obstruction is easily remedied by excising the web, if necessary leaving a flap of skin to cover the raw area and thereby prevent stenosis.

### *Congenital Occlusion of the Posterior Nares*

This condition is also uncommon. Here one or both choanæ may be obstructed by a bony diaphragm. This diaphragm represents the vestigial remains of the bucco nasal membrane. The chief symptom noticed is cyanotic attacks at or shortly after birth accompanied by great difficulty in sucking (see p 42).

The treatment is obviously removal under general anæsthesia. The diaphragm is broken through with a burr or gouge with a finger in the nasopharynx acting as a guide and guard. The edges together with a moderate sized piece of the edge of the septum are then punched away with a sphenoidal forceps.

### Dermoid or Hairy Polypus of the Nasopharynx

This is a rare nasopharyngeal tumour which usually causes symptoms during the first few days of life but which has in a few cases remained undiagnosed until adult life. The growth is usually pedunculated and of fleshy consistency and may be long and club shaped or comparatively sessile. It is covered by skin bearing sebaceous and sweat glands sometimes containing plain muscle fibres and covered with fine downy hairs. Other tissues such as cartilage, serous and mucous glands and lymph follicles have also been found. The pedicle is usually attached to the nasopharynx.

In most cases the growth causes attacks of dyspnoea and cyanosis with difficulty in sucking and swallowing soon after birth. If it is of the long pedunculated type the chain of symptoms is typical. After an alarming attack of choking the infant makes a violent expiratory effort or vomits and considerable relief ensues. On examination a fleshy club shaped growth is seen in the buccal cavity or protruding through the mouth. The tumour gradually slips back into the pharynx and causes another choking attack by its ball valve action at the *introitus laryngis*. If however the tumour is more sessile in shape it may merely occlude the nasopharynx. Here the growth will bulge the soft palate forwards and may present below its free border. In this case the symptoms will be less paroxysmal in character and will resemble those caused by adenoids or occlusion of the choanae from other causes.

Dermoid polypi have to be distinguished from teratomata and from mixed tumours. Treatment consists in removal with a snare passed through the nose and presents no difficulty in skilled hands.

### Congenital Laryngeal Stenosis

This is a very rare condition in which a diaphragm partly occludes the larynx. Inspiratory stridor with dyspnoea on

exertion is noticed soon after birth. The crying and speaking voice is hoarse and weak. Usually the condition is mistaken for congenital laryngeal stridor on account of the difficulty in seeing the anterior commissure by indirect laryngoscopy in children and the correct diagnosis is not made until later in life. On direct laryngoscopy a crescentic rounded web is seen occupying the anterior commissure. On phonation this web folds between the cords causing the harsh weak voice.

Treatment is best avoided if possible. If it is necessitated by respiratory embarrassment the galvano-cautery is probably the best means of dividing the web. It may be necessary to introduce an intubation tube to prevent cicatricial stenosis following.

### Congenital Stenosis of the Œsophagus

This is a rare condition in which there is failure of complete fusion of the œsophagus and stomach and persistence of that part of the diaphragm separating them which should normally disappear during development (St. Clair Thompson). The chief symptom is regurgitation of food and inability to take solids. The condition is most often mistaken for spasmodic stricture and patients may subsist on fluids until adult life is reached.

On œsophagoscopy a valvular flap is seen at the cardia guarding a pinhole orifice. This distinguishes the defect from the star-shaped lumen of spasmodic stricture and from the crescentic shape of the stenosed thoroughfare caused by external compression (St. Clair Thompson).

Treatment is dilatation by per oral endoscopy or digitally from the stomach.

### Congenital Imperforation of the Œsophagus

This is a rare condition but is perhaps not so uncommon as is supposed the diagnosis probably being often missed. Two forms occur. In one the middle third of the gullet is represented by a fibrous cord. In the other the upper third of the œsophagus ends blindly and is connected with the lower third by a fibrous cord the lower third being patent and opening directly into the trachea or one of the main bronchi.

The symptoms are typical. The child is born strong and vigorous and suckles readily. The food however, is immediately

rejected. In the second form gastric juice also escapes into the air passages through the lower third of the œsophagus when vomiting occurs and causes desperate choking and asphyxial attacks. Screening an opaque meal will settle the diagnosis.

The child usually lives about a week and treatment is of no avail. Gastrostomy has always failed and is unjustifiable.

## CHAPTER XXXV

T. G. WILSON

### DISEASES OF THE NOSE PHARYNX AND LARYNX

(Nasal Obstruction and Discharge in Infants Treatment—Retropharyngeal Abscess Treatment—Pathological Enlargement of the Thymus—The Larynx Simple Laryngitis Laryngitis Stridulosa Laryngismus Stridulus Congenital Laryngeal Stridor )

#### Nasal Obstruction and Discharge in Infants

WHEN occurring at or soon after birth this may be due to one of several causes of which the most important is congenital syphilis

Injuries to the nose from compression by a blade of the forceps during delivery are probably more common than is supposed and in addition to producing obstructive symptoms soon after birth may play a large part in the etiology of the deflected septum of later life Other causes are acute coryza birth injuries and occasionally congenital adenoids

#### Congenital Syphilis

This subject is fully dealt with elsewhere (see p 234) It is necessary however to discuss briefly here nasal obstruction in the new born due to the disease

The first symptom is snuffles or catarrhal blockage of the nasal cavities accompanied by a thin glairy rhinorrhœa This appears about the sixth week and is progressive the discharge becoming thick and purulent and being associated with excoriation of the nares and crust formation and macular patches in the pharynx and larynx

The differential diagnosis from congenital adenoids and chronic catarrh is often difficult and a word of warning to the student is necessary here Congenital syphilis is a rare disease and is rarely seen except in special clinics Minor degrees of nasal catarrh are common Hence no suggestion of even the possibility of congenital syphilis should be made to the parents till further confirmation is obtained Serological tests

together with a general examination of the child will almost always settle the matter for syphilitic snuffles are almost always associated with other manifestations of the disease. It may indeed be said that syphilis will never be the cause of snuffles in an otherwise healthy baby who is gaining weight normally.

**Treatment** The treatment of congenital syphilis does not concern us here. Acute coryza is helped by the instillation of liquid paraffin with or without the addition of a little menthol (gr v ad oz i). It has been suggested that this can be well applied by means of a catheter dipped in the solution and passed through the nasal cavities as far as the nasopharynx. Adenoids when present are removed. This trifling operation as a rule does not in the least disturb the patient's equilibrium and is often performed without anaesthesia.

### Retropharyngeal Abscess

Retropharyngeal abscess may be either acute or chronic. The acute pyogenic type is that which is most often found in infants. It is very important clinically because the diagnosis is often missed and if surgical intervention is not undertaken the patient usually dies. Any of the acute infectious fevers—measles, scarlatina and diphtheria—may be the immediate cause, but the streptococcus is usually the infecting organism. An acute abscess may occur as early as the second week of life. The chronic form is due either to tuberculous cervical caries or tubercular glands.

Suppuration takes place in the space between the posterior pharyngeal wall and the pre-vertebral fascia. These structures are adherent in the middle line but separate laterally to enclose a chain of lymph glands on either side. Infection reaching these glands *via* the tonsils or adenoids is the usual cause of the condition.

Symptoms may be very slight at first and the pyrexia, dysphagia and general malaise may be attributed to some other cause until the abscess has become large enough to cause dyspnoea and difficulty in swallowing. The larynx will become obstructed early in the disease by pressure or by spreading œdema and a hoarse croupy cough may soon develop. The child becomes restless, sleep is disturbed and food is refused. The child may produce a snoring noise even when the nostrils are closed. Crying is thick and throaty and respiratory

embarrassment may be marked. The mouth remains open and dribbles and the glands on the affected side are usually enlarged and tender. One important sign of considerable help in diagnosis is that the head and neck are held stiff and rigid, the head being usually inclined to the healthy side.

On examination the diagnosis is readily made. A rounded, shining swelling is seen to occupy one or other side of the pharynx, sometimes pushing forwards the posterior faucial pillar.

It is important to keep the head in the mid line during inspection, as if turned sideways the transverse process of the axis normally causes a bulge to appear at the side of the pharynx. Palpation is a valuable aid and should always be carried out. Sometimes fluctuation will be found more often a boggy, phlegmonous swelling. In view of the undoubtedly large number of cases in which the diagnosis is missed the importance of palpating the pharynx in all cases of dyspnoea or dysphagia in infants must be stressed.

**Treatment.** Treatment consists in making an incision through the mouth as soon as the diagnosis is made whether fluctuation is present or not. With the left forefinger as a guide, a guarded scalpel is introduced and a vertical cut made in the most prominent part of the swelling. As soon as the incision has been made the child is turned over to prevent aspiration of blood and pus. No anæsthetic is necessary or advisable.

### Pathological Enlargement of the Thymus

This condition has long been a subject of controversy. The enlargement of the thymus recognised by most authorities as a pathological condition which may produce sudden death at any moment of stress, but apparently most often during anæsthesia, is probably a myth. During health the thymus is normally large, but it becomes small and involuted as a result of inanition or wasting illnesses. This small involuted thymus is, of course, most often found at autopsy, and has come to be mistakenly regarded as the normal gland.

Two views are held as to the cause of thymic deaths. The first explains them as being due to mechanical obstruction, the second by the occurrence of the status thymo-lymphaticus. This is a systemic disease affecting not only the thymus but also other lymphoid tissue such as the lymph glands, Waldeyer's

ring Peyer's patches and the intestinal nodes, and is associated with hypoplasia of the cardiovascular system and probably also of the chromaffin system and gonadal glands. The child who exhibits this condition has a distinctive body constitution later in life whereas the child with a simple hypertrophied thymus does not differ from others except that it is usually plump and well nourished.

In cases of upper respiratory obstruction all other possible causes of stridor should be excluded before a definite diagnosis is made. Direct inspection of the larynx should always be carried out and this procedure will often result in a diagnosis of congenital laryngeal stridor.

### THE LARYNX

There are several important differences between the infantile and the adult larynx.

(1) In the child the larynx is smaller in comparison to the rest of the body than is the case in the adult. Its lumen is therefore smaller in comparison to the body as a whole than in later life and anything which tends to cause obstruction is likely to produce alarming and acute dyspnoea.

(2) The cartilaginous skeleton of the larynx becomes harder as life progresses and in elderly male subjects is usually almost completely ossified. In infancy the cartilages are soft and pliable and the submucous tissue is loose and vascular. When inflammation occurs in childhood the attendant swelling is relatively greater than in adult life and urgent obstructive symptoms occur more readily. In adult laryngitis obstructive symptoms seldom arise and when they do are usually the result of inflammatory swelling rather than of spasm.

(3) The nervous system in infants is unstable and the larynx is particularly sensitive and liable to spasm.

These considerations are of importance in all laryngeal affections of children and are well exemplified in

#### Simple Acute Laryngitis, or Croup

**Ætiology** Naso-pharyngeal catarrh due to adenoids is perhaps the most common predisposing cause. The condition may also be seen during the invasion period of an acute

infectious fever particularly measles and whooping cough or may be brought on by prolonged crying

**Pathology** The pathology is that of any catarrhal inflammation. At first there is redness and swelling due to vascular engorgement of the mucous and submucous layers followed by increased flow of mucus and perhaps shedding of the surface epithelium. Resolution usually follows in a few days but the inflammation may spread downwards to the trachea and bronchi, and pneumonia may follow.

**Symptoms** Symptoms may occur with alarming rapidity in the case of small children. Some slight alteration in the voice and perhaps a little cough may fail to draw attention and a few hours later the child may fall ill with a temperature as high as  $104^{\circ}\text{F}$  and a hoarse croupy cry. Later alarming obstructive symptoms may ensue with great cyanosis and respiratory distress. There is usually considerable inspiratory stridor, and the epiglottis and infra sternal spaces may be sucked in with each inspiration. If the larynx is examined during the attack the mucosa is seen to be red and engorged with the infolded infantile epiglottis somewhat swollen and obstructing the view of the *introlitus laryngis*. No membrane is seen but the mucous membrane of the infra glottic region will be seen to be red and swollen so that the lumen of the trachea is reduced to an antero posterior chink. A few rales may be heard in the chest, and some bronchial catarrh usually persists a few days after the attack. This is the disease which is often known simply as 'croup'.

When the spasmodic element predominates the condition is sometimes called *laryngitis stridulosa*—a subdivision of the disease which we consider unnecessary and misleading. In this type of case the croupy attacks occur more suddenly and subside with equal rapidity. The voice may be normal a few hours before the attack, which is usually nocturnal and a high temperature is uncommon. The voice may rapidly return to normal but bronchial catarrh may persist for a few days. An injection of adrenalin is particularly helpful in this type of infantile laryngitis, which is often a precursor of asthma in later years.

**Diagnosis** Acute laryngitis in infants differs from diphtheritic laryngitis by its sudden often nocturnal onset, by the stronger voice and croupy cough. In laryngeal diphtheria the onset is often insidious, the cough is more feeble, and the

disease is progressive and not subject to variations in the severity of the symptoms. A throat swab should be examined in every case and if a membrane is present anti diphtheritic serum should be administered. Diphtheria is not common in the first year of life although we have seen it as early as the third month and again in twins of six months.

**Treatment** During the attack hot vapour from a steam kettle is useful and a hot mustard bath may cut short the attack. Hot fomentations or poultices applied externally to the larynx are said to be useful. A drachm of 1 in *Ipecacuanha* as an emetic may help to clear the larynx of mucous secretions and thereby give relief. Two or three drops of adrenalin 1 in 3 000 will ease the spasm rapidly particularly in the type of case described as *laryngitis stridulosa*. Tracheotomy may occasionally be required and the necessary instruments should be at hand particularly when pallor and restlessness follow cyanosis. After the acute stage has subsided the child will require rest in bed for a few days and later it is most important that abnormalities of the upper respiratory tract should be attended to and in particular that enlarged tonsils and adenoids should be removed.

### Laryngismus Stridulus

**Laryngismus stridulus** is a form of stridor which comes on suddenly usually in male infants over three months of age and is not accompanied by fever. It is now considered to be a complication of tetany and is often associated with rickets, gastro-enteritis, bronchitis, indigestion and enlarged tonsils and adenoids.

**Symptoms** The symptoms which may be very alarming are those of inspiratory obstruction. There is no difficulty in expiration. The child stops breathing, becomes blue in the face and goes into a condition of semi asphyxia which may last from half to one and a half minutes. The pupils dilate, the arms are thrown about, the chest heaves and all the accessory muscles of respiration are called into play. The lower ribs are sucked in and convulsions and incontinence of urine and feces may occur. With all this the child rapidly becomes terrified. After about a minute relief takes place usually heralded by a long deep breath but death may occur in rare cases.

There is no rule as to recurrence. Another attack may occur

shortly afterwards, and be repeated frequently, or the symptoms may not come on again for a considerable period

If the larynx is examined, no abnormality is found

**Pathology.** These symptoms have been ascribed to "spasm" of the larynx, caused by disease of the nervous system. It is, however, more usually considered to be caused by collapse of the laryngeal soft tissues out of sheer feebleness (Vivian Poore, *Medical Chronicle*, 1898). The majority of these patients are debilitated, and in many adenoids are present. Although the child breathes through the mouth in the daytime at night the instinct of nasal respiration is so strong that the mouth is closed. As a consequence of the partial nasal blockage the blood becomes insufficiently oxygenated. "At last the child becomes semi-asphyxiated and to remedy this has to take a sudden deep breath. The feeble larynx fails to open because the weak posterior crico-arytenoid muscles do not contract with sufficient strength to open the glottis, and the soft parts are sucked together by the rushing air current. As the air imprisoned in the lungs is absorbed, the blood becomes more and more charged with  $\text{CO}_2$  until finally the larynx opens and air rushes through and the characteristic 'crow' is emitted" (St Clair Thompson)

**Diagnosis.** The salient features of the condition are —

- (1) The sudden onset
- (2) Equally rapid and complete subsidence
- (3) The normal voice between the attacks
- (4) Absence of fever

These points are sufficient to distinguish between laryngismus and diphtheria or acute laryngitis. Congenital laryngeal stridor is differentiated by the fact that it occurs in strong healthy children and by other features (see diagnostic table)

**Treatment.** Treatment may be divided into —

- (1) Immediate
- (2) Prophylactic

While the acute attack is in progress the child should be supported in the sitting up position, and a free supply of fresh air ensured. Cold compresses to the chest, smelling salts, and rhythmical traction of the tongue (St Clair Thompson) may be tried. It is best to be prepared for tracheotomy, although this is seldom required. In acute cases the associated alkalosis should be treated by the administration of calcium chloride by mouth.

Between the attacks the fundamental principle in treatment is the correction of the causative factor tetany. Adjustment of diet with ample supply of vitamin D is essential. General debility must be combated by suitable measures. The tonsils and adenoids should be removed only if definitely enlarged or infected. The bowels should be regulated and the functions of the skin promoted by freedom from excessive clothing and plenty of fresh air and exercise.

### Congenital Laryngeal Stridor

This is a rare disease which commences soon after birth and generally disappears during the second year.

**Ætiology.** The most interesting point from the diagnostic point of view is that it occurs usually in robust infants. This alone differentiates the disease from laryngismus stridulus for which however it should not be mistaken. Examination by direct laryngoscopy has demonstrated that the cause is the presence of an exaggerated infantile type of larynx. The epiglottis is very long and rolled so that its lateral margins meet posteriorly and form an almost complete cylinder above. The ary-epiglottic folds are in consequence closely approximated leaving a very small air way. The croaking noise is caused by the unsupported laryngeal walls and the loose tissue on the summits of the arytenoids which vibrate to and fro during inspiration. Gabriel Tucker (*J A M A* 99 1899 1932) states that in the infant the larynx is at an angle from behind forwards and downwards towards the glottic lumen. With descent of the larynx as a whole *e.g.* when the child cries the epiglottis assumes a more nearly vertical position making the axis of the lumen at the entrance to the larynx more nearly in line with the subglottic larynx and trachea. An increase in this angle of entrance into the larynx may become one of the factors in the production of so called congenital stridor. A subglottic diameter of 4 mm. in an otherwise normal child should be considered as congenital stenosis.

**Symptoms.** The outstanding feature is *inspiratory* stridor usually noticed soon after birth. It has been variously described as clucking, purring, grunting or croaking. Inspiration commences with a croaking noise and ends with a high pitched noise. Expiration is accompanied by a short creak when the stridor is loud but at the other times it is noiseless. (John

Table VIII

	Type of child	Age	History	Onset	Progress	Voice	Temperature
Simple acute laryngitis	Variable	1 from 11 to 20 months	Catarrhal infection	Sudden	Fluctuating severely	Strong and hoarse	Raised
Diphtheria	Variable	Not usual in first year	Klebsiella or diphtheria	Insidious	Progressive	Weak	Raised
Laryngostridor	Weak, puny	1 from three months	Pituitary	Sudden	Free of symptoms between attacks	Normal between attacks	Normal
Congenital laryngostridor	Usually not	1 from birth	Congenital stenosis		Free of symptoms between attacks	Normal between attacks	Normal

Thompson) The loudness of the stridor is proportionate to the depth of the breathing. Any stimulation, such as a change from a warm to a cold atmosphere, a fright, or sudden shock, may bring on or increase the stridor. The cry is not in the least hoarse. Abdominal and lower rib retraction are fairly usual but cyanosis is seldom seen. There are usually periods of complete freedom from symptoms.

**Progress** The disease may get worse for a few months after birth but after this the stridor usually diminishes, and almost invariably disappears completely by the third year, with the development of the larynx. The prognosis is favourable, and any danger to life is usually due to lung complications.

**Treatment** Treatment is directed to maintaining the general condition. Tracheotomy should only be performed as a last resort as it has been attended with a high mortality in these cases.

## CHAPTER XXXVI

T G WILSON

### ACUTE OTITIS MEDIA

(The Middle Ear, Mastoid Process, Pneumatic Mastoid, Diploëtic Type, Sclerotic Type—The Infantile Mastoid, External Auditory Meatus Tympanic Ring, Method of Examination—Acute Otitis Media in Infants—Simple Suppurative Otitis Media—Parenteral Otitis Media—Tuberculosis—Bacteriology, Treatment, Complications, Operation)

BEFORE describing the *ætiology* of acute suppurative otitis media in infants it is necessary to consider the infant's skull, so that we may appreciate the difference between the infantile ear and the adult organ

#### The Middle Ear

The middle ear is composed of three parts (1) the Eustachian tube, (2) the tympanic cavity proper, and (3) the mastoid process. These three form an air-containing cleft running upwards, outwards and backwards from the nasopharynx. The tympanic cavity does not differ greatly from the adult type. It roughly resembles a quadrangular biconcave lens (Portier). Its height and width are about 1.4 mm. and its depth varies from 2 to 6 mm. It is crossed by the chain of ossicles which connect the drum or tympanic membrane with the internal ear. The ossicles are very nearly the same size in infancy as in adult life, and the same may be said of the tympanic cavity as a whole.

The other two parts of the middle ear cleft are, however, very different in infancy. The adult Eustachian tube is from 31 to 38 mm. long and consists of two parts: (1) the osseous and (2) the membrano-cartilaginous portion. The two parts of the tube are not quite in the same straight line, but make an obtuse angle at the junction. The general direction is forwards, inwards and downwards from the tympanic cavity. At the isthmus, where the bony and cartilaginous portions join, the lumen is about 2 mm., but it is considerably expanded at either end. It is lined with columnar ciliated epithelium.

In the new born infant the Eustachian tube presents marked differences to the adult type

(1) It is very much shorter (14-15 mm )

(2) The tympanic orifice and the calibre of the tube are quite as large as in the adult. The whole canal is therefore much wider relatively than the adult

(3) The two portions of the canal are very nearly in the same straight line

(4) The direction of the canal is very nearly horizontal the pharyngeal opening being therefore at the same level as the tympanic orifice whereas in the adult it is 15 mm lower

(5) The pharyngeal mouth of the tube is on a level with the hard palate. In the adult it is at least 10 mm above this

These differences in type between the infantile and the adult Eustachian tube are of extreme practical importance and demonstrate how easy drainage of the middle ear should be in infants provided that there is no obstruction in the naso-pharynx. We should notice however that the horizontal direction of the tube is against efficient drainage

### The Mastoid Process

The adult mastoid process is a conical mass of bone which projects downwards behind the bony meatus. It consists of an outer shell of dense bone enclosing a central space filled with bony cells which contain air or spongy tissue. The largest of these air spaces lies at the upper anterior part of the mastoid process. It is known as the mastoid antrum and communicates with the tympanic cavity by means of an opening called the *aditus ad antrum*.

Three types of adult mastoid process are recognised according to the amount of cellular development which has taken place —

(1) *The Pneumatic Mastoid*. Here the interior is divided into a number of cellular spaces each lined with mucous membrane

(2) *The Diploetic Type*. In this type the interior of the mastoid is filled with a vascular spongy tissue, somewhat resembling the tissue which occupies the space between the diploe of the cranial bones

(3) *The Sclerotic Type*. Here the mastoid process has practically no cells and consists almost entirely of ivory bone

There has been considerable controversy as to the causation of these three types of mastoid. Wittmaack believes that the sclerotic or acellular mastoid is a result of pre-natal or infantile inflammation. Owing to infection the vitality of the mucous membrane is damaged and normal pneumatization does not take place. The diploetic mastoid is a minor degree of the same condition, the cellular mastoid being the normal. Chertle however considers that the three types of mastoid are different anatomical types and the weight of evidence appears to be in his favour. The frequency of suppuration in sclerotic bones is, in his opinion, a consequence rather than a cause of the bony sclerosis.

### The Infantile Mastoid

*At birth the infant strictly speaking has no mastoid process.* There is one cell only, the mastoid antrum, and the lower line of the mastoid process is level with the bottom of the meatus. As development proceeds cells bud out in a downward direction and by the fourth year the mastoid is developed almost to its adult form.

### The External Auditory Meatus

The external auditory meatus presents marked differences in the adult and the new born. In order to gain a clear view of the anatomy it is necessary to briefly recapitulate the development of the temporal bone after birth.

*At birth the temporal bone consists of three distinct parts—the petro-mastoid, the squamo-zygomatic, and the tympanic ring.*

The *tympanic ring* is a small ring of bone deficient at the upper and anterior part for one eighth of its circumference. A sulcus is present inside the ring for the attachment of the drum membrane. The tympanic ring is applied against the petrous bone closing with the tympanic membrane, the lower and outer half of the tympanic cavity. Osseous union takes place during the first year of life. The deficient eighth part of the circumference is supplied by the squamo-zygomatic which also forms the roof of the tympanic cavity.

The external auditory meatus and drum membrane of the infant at term presents the following differences from the adult type. (1) *There is no bony meatus.* The drum membrane is

therefore not protected by being at the bottom of a bony canal but lies in the same plane as the outer and under surface of the skull. Its position is much more nearly horizontal than in the adult. (2) The entire canal is membrano-cartilaginous. Its direction is outwards and upwards so that the drum membrane and the roof of the canal are almost in the same plane.

**Method of Examination** These anatomical differences give rise to certain difficulties in examination.

As the lumen of the external auditory meatus in children is very small it is sometimes difficult to see the drum. An electric auriscope is of very little use in the case of infants up to three months. A lead mirror and reflected light give a much better view of the drum. A very small speculum should be used and the pinna should be pulled *downwards* not upwards and outwards as in the case of adults. The drum is almost as large in the infant as in the adult and a full view of it can be obtained only by moving the speculum about.

On account of the comparatively wide and short Eustachian tube of young children air easily enters the tympanum and a bulging and slightly injected drum does not therefore always mean that pus is present and is certainly not an indication for paracentesis. If on examination details of the drum can be made out and especially if the short process and handle of the malleus and the light reflex can be seen immediate paracentesis is not necessary. The light reflex is the most important of these landmarks as it is the first to disappear in the presence of inflammation. If the membrane is injected it should be watched and if the inflammation is increasing paracentesis should be done without hesitation.

## ACUTE OTITIS MEDIA IN INFANTS

Acute otitis media in infants may be classified in three groups —

### (1) Simple Acute Suppurative Otitis Media

The corresponding disease to acute suppurative otitis in adults but with essential differences due to the age of the patient. This may again be sub-divided into two groups (a) *obvious* and (b) *latent*.

## (2) Parenteral Otitis Media

or otitis media in association with diarrhoea and vomiting in infants

## (3) Tuberculous Otitis Media

## (1) Simple Acute Suppurative Otitis Media

**Ætiology** There is no doubt that the greater incidence of otitis media in infants compared with adults is due, in the main, to two causes (1) the greater susceptibility of children to catarrhal infections, and (2) the frequent presence of enlarged "adenoids." Children are particularly liable to infections of the mucous membranes which when they occur are more likely to lead to pyogenic inflammation than in the case of adults. This is confirmed by the fact that in the majority of infants suffering from acute otitis media enlarged cervical glands are to be found, suggesting the presence of pharyngitis.

Enlarged nasopharyngeal adenoids are, of course a very important ætiological factor. The tonsils do not often give rise to trouble during the first year of life and seldom become seriously infected before the third or fourth year. Adenoids, however, are often present at birth, and constantly require removal during the first year. They give rise to trouble in two ways (a) by becoming infected and passing on the infection to the middle ear, and (b) by mechanically blocking the Eustachian tubes, thereby preventing drainage of the middle ear cleft. The short, wide Eustachian tube of the infant provides a ready path for infection of the tympanum but allows drainage equally readily, provided that there are no obstructing adenoids.

Colds are therefore a common cause of acute otitis media in infants. The acute fevers—scarlatina, measles and the like—do not play so important a role in the first couple of years of life as they do later. Measles and scarlatina, however, may both produce a very serious type of otitis, and scarlatina in particular may cause considerable destruction of the drum and ossicles in a very short time. Nearly every case of mumps is accompanied by some redness of the drum. This is easily explained by the fact that there is in children up to five or six years of age an opening in the lower part of the meatus, the dehiscence in the tympanic ring articulation, which is in almost direct contact

with the inflamed parotid. Sometimes this inflammation passes to the labyrinth and causes severe permanent deafness very often without formation of pus.

It is probable though difficult to prove that otitis media and mastoiditis are caused by infection through the blood stream more often than in adults.

### (1) (a) Obvious Acute Suppurative Otitis Media

The symptoms vary greatly according to the severity of the infection. Usually nurslings are restless for several hours older infants roll their heads from side to side and put their hands up to their ears. These signs may be caused by reflex otalgia from teething pains but if the temperature is high  $103^{\circ}\text{F}$  to  $105^{\circ}\text{F}$  the ears are probably the cause. Vomiting is usual and meningismus may be present.

### (1) (b) Latent Acute Suppurative Otitis Media

A great deal has been written and said recently about latent otitis media in children. Some authorities classify it under the heading of parenteral otitis others deny its entire existence.

Every doctor is familiar with mild cases of infantile otitis media in which there may be little or no constitutional disturbance. The child perhaps cries a little or has a disturbed night and a few hours later a discharge is noticed in the meatus. Here the infection is usually a mild one and the ear rapidly dries up unless adenoids are present. This is not properly speaking latent otitis media but merely a mild form of the classical disease. There is however a dangerous type of otitis media which undoubtedly does occur and which deserves the title of latent otitis media. Here constitutional symptoms are severe and in the absence of symptoms pointing to disease in any other quarter the ears are examined. The drums however are normal or nearly so and even if paracentesis is done very little pus is exuded from the middle ear. The symptoms continue to be severe and the temperature and pulse may rise very high even up to  $105^{\circ}\text{F}$ . Reflex vomiting may occur. If at this stage the mastoid antrum is opened it will be found to be full of pus. With prompt operation immediate recovery is probable.

This condition is, in all probability, a blood-borne infection, and often primary in the mastoid process

*It clearly follows here that in the presence of unexplained pyrexia, every infant should have its ears examined. The great majority of cases will prove to be normal, but a number of lives will be saved which might otherwise be lost*

**Bacteriology** More than half the cases of otitis media in infants appear to be due to infection by the pneumococcus. The dreaded type III infection, by the so-called streptococcus mucosus, is said to be particularly common in infants.

**Treatment.** Early paracentesis should always be undertaken. Generalised injection of the tympanic membrane, with, in particular, loss of the light reflex, is the indication. On the first day, the thin blood-stained discharge should be mopped away with sterile cotton wool pledgets as often as is necessary. On the second day, and subsequently the meatus is thoroughly cleansed three hourly with a mild alkaline lotion and antiseptic drops instilled. The type of drops used is unimportant, except that glycerine of carbolic acid and peroxide of hydrogen both macerate the epithelium and are unsuitable for use in the presence of a purulent discharge. Fifty per cent spirit argyrol or mercurochrome are perhaps most commonly used.

Adenoids, when present, should be removed in a week or so when the acute stage has passed. Complications should be treated as they arise.

**Complications** (1) *Mastoiditis* Since the mastoid process does not exist in the first few months of life, mastoiditis would appear to be a paradoxical term. Mastoid antritis is more correct. Since, however, retro auricular periosteal abscess and even necrosis of the outer wall of the mastoid process are often seen and bear a very similar clinical appearance to certain forms of mastoiditis which occur in later life, it is convenient to refer to it by the same name.

Mastoiditis, resulting from obvious, latent or parenteral infection, does not differ in type, except that in the latter varieties it is more usually bilateral.

There are certain differences between the symptoms of mastoiditis in infancy and in adult life.

(1) The infant cannot, of course, differentiate between antrum pain and pain due to pus in the tympanum. The type of pain present is, therefore, of no diagnostic value.

(2) Fever is usually a well marked symptom of otitis media.

in children and should it fall any slight exacerbation of the tympanic condition will bring about a sharp rise. Fever is therefore of little assistance and is a less important symptom than in the adult.

(3) Symptoms suggestive of cerebral irritation or infection are also of less importance than in the adult as the cerebral centres are more easily excited by peripheral irritation. Sudden chills convulsions hyperpyrexia vomiting etc. are also of less significance than in the adult.

(4) Petro auricular swelling with perforation of the cortex and periosteal abscess causing forward displacement of the pinna (in fact what was once looked upon as typical mastoiditis) is much more common in children than in adults. This is because the outer mastoid cortex or antrum wall is much softer and more spongy than in adults.

When dealing with simple otitis media in infants operation is seldom called for in the absence of this retro auricular œdema. There is only one cell to deal with the mastoid antrum which is able to discharge its purulent contents into the external auditory meatus or Eustachian tube & the middle ear. If this drainage is not satisfactory removal of adenoids will usually make it so. Operation may be reserved for those rare cases in which the discharge does not clear up within four to six weeks of the removal of adenoids. In cases of parenteral otitis however the position is different. Here the discharge will not dry up until the bowel condition is better and it is better to operate and thereby drain the middle ear cleft from behind if the condition is not improving.

**Operation.** The operation in young infants is simple. An incision less than 1 inch long is made in the retro auricular groove and the outer cortex of the mastoid is removed disclosing the mastoid antrum which is drained with a rubber tube. In slightly older children more cellular development has taken place and infected cells are dealt with. The whole procedure need not take more than a couple of minutes. Some surgeons use local the majority light general anaesthesia.

Of the intracranial complications *meningitis* is by far the most common and occurs relatively more often than in the adult. *Extradural abscess* *brain abscess* and *sinus thrombosis* occur but less often than in older children and adults. This is surprising as the open sutures and soft bones would appear to favour extension.

## Otitic Hydrocephalus

A rare intracranial complication which occurs is *otitic hydrocephalus*. This occurs most often in children and adolescents, and has been defined as a state of increased intracranial pressure occurring in association with otitis media and due to the presence of an excess of normal cerebrospinal fluid. The condition has been recognised for some time but its pathology remains obscure. There is no evidence as to whether it is due to increased secretion by the choroid plexus or to defective absorption through the arachnoid villi. It may be due to an obstructive internal hydrocephalus caused by occlusion of the efferent channel from the ventricles. The most constant symptom in older children and adults is well marked papill oedema sometimes with headache. Papilloedema is seldom seen in infants since any increase in intracranial tension is accommodated by expansion of the whole head. This is allowed for by the soft cranial bones and open sutures. Nausea and vomiting may occur but the temperature and pulse remain normal. In some cases meningeal symptoms are present. Convulsive seizures may occur.

Treatment consists in lumbar puncture. The cerebrospinal fluid withdrawn may be under considerable pressure (300 mm) and of considerably increased volume. If this does not relieve the symptoms ventricular puncture should be undertaken.

It has been said that intracranial complications are comparatively rare in infants. When such complications do occur, their treatment does not differ in essentials from that performed in adults.

## (2) Parenteral Otitis

**Ætiology** There is no doubt that otitis media is a very serious complication of infantile diarrhoea and vomiting. Recent statistics show that when it occurs the prognosis is very unfavourably affected. In one series death took place in 30 per cent of cases without otitis and in 50 per cent of cases when otitis was present. In other words the death rate is almost doubled when the ears are infected.

It must be remembered however, that post mortem examination will show pus in the middle ears of almost all infants no matter what the cause of death. This would appear to show that the infection is an 'agonal' condition. In some cases

there are only redness and congestion of the tympanum, in others suppuration throughout the entire middle ear cleft

There has been considerable controversy as to the exact role played by the otitis media. Laurence performed autopsies on children who had died as the result of diarrhoea and vomiting, and in some cases found no bowel abnormality, but pus in the middle ear. He therefore concluded that the otitis media was the cause of the gastro-enteritis, and with this view Le Mee and other authors agree. Mollison considers the ear condition to be a form of latent otitis, and looks upon both the otitis media and the enteritis as a reaction to the same infection. He stresses the well known liability of infants and children to infection of the mucous membranes.

Krassnig on the other hand argues that intestinal disturbance is a cause of otitis media. If the bowel infection does not clear up, otitis media of a mild type develops which clears up readily if the intestine recovers. He argues that in bronchitis much pus is swallowed with the sputum, but enteritis does not follow.

It will be noted in hospital practice that cases of enteritis admitted soon after the onset of the disease usually have normal ears on admission but they often develop otorrhoea after some days. On the other hand cases admitted in an advanced stage often have otorrhoea on admission. The explanation is not far to seek. Infants in this condition are often, perhaps usually, left lying on their backs in their cots. When vomiting takes place the child is too weak to turn over and properly evacuate the regurgitated gastric content. In consequence a certain amount lies in the nasopharynx, and presumably may pass down the straight wide Eustachian tube of the infant into the middle ear. This is the more easy when we remember that the Eustachian tube is relatively much lower down in the infant than in the adult being in fact opposite the hard palate. It is also supported by the fact that the nasopharyngeal end of the Eustachian tube is opened during deglutition by the action of *tensor veli palatini* and other muscles. It is now recognised that infants, no matter how seriously ill, should never be fed in the supine condition. Milk and half-digested food form excellent pabulum for bacteria. It is easy to understand that the more severe the gastro-intestinal infection the weaker the child is, and, consequently, the more likely it is for the ears to become infected in the manner described. This explains why otitis media is a com-

plication of such bad omen in gastro enteritis. It is an index of the weakness of the child and of the degree of toxæmia rather than a fatal complication in itself.

Bacteriology supports this view since the greater number of these cases are found to bear a mixed infection.

Parenteral otitis is therefore what might be called an hypostatic infection. This explains the insidious onset of the aural disease and why a low grade infection is so often found. Older children with diarrhoea and vomiting do not get otitis as they are not so weak that they cannot turn over and evacuate the vomit from their nasopharynx.

**Treatment.** The treatment of parenteral otitis is that of simple otitis media in infants with one very important exception. If the discharge does not dry up early antrotomy should be carried out. The ear will not get well until the bowel does. If the patient is not recovering it may be because the otitis media has set up a vicious circle and is retarding the patient's general condition. The simple operation of antrotomy should therefore be performed and the middle ear thereby drained from behind.

### (3) Tuberculous Otitis Media

Tuberculous otitis media is said to be common in infants. This is probably incorrect. It occurs primarily in the mastoid process thereby differing from tuberculous otitis media in the adult which is always secondary to tuberculosis elsewhere in the body. The infection is usually from milk but it may also occur in breast fed children by maternal infection.

In the early stages the disease may not differ greatly from simple otitis media but a sign of great importance is enlargement of the pre auricular gland. This is a danger signal the significance of which should not be missed. In the later stages the chief clinical feature is great bony destruction with the formation of sequestra and but little pus. The facial nerve is usually involved early and the whole labyrinth may separate as a sequestrum. The meninges are usually involved and a fatal termination is the rule. A thorough radical mastoid operation may effect a cure.

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